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CONTENTS OF VOLUME XIV

JULY, 1917. NUMBER 1

PAGE

EDITORIAL: PROTECTION OF CHILD LIFE DURING THE WAR 1

FOCAL LESIONS PRODUCED IN THE RABBIT BY COLON BACILLI ISOLATED FROM PYELOCYSTITIS CASES. HENRY F. HELMHOLZ, M.D., AND CAROL BEELER, CHICAGO 5

TWENTY-FOUR-HOUR METABOLISM OF TWO NORMAL INFANTS WITH SPECIAL REFERENCE TO THE TOTAL ENERGY REQUIREMENTS OF INFANTS. FRITZ B. TALBOT, M.D., BOSTON 25

SOME ANALYSES OF VEGETABLES SHOWING THE EFFECT OF THE METHOD OF COOKING. ANGELIA M. COURTNEY, HELEN L. FALES AND FREDERIC H. BARTLETT, M.D., NEW YORK 34

ENLARGEMENT OF THE THYMUS TREATED BY THE ROENTGEN RAY. ALFRED FRIEDLANDER, M.D., CINCINNATI 40

THE VALUE OF THE VON PIRQUET TEST AS CONTROLLED BY NECROPSY FINDINGS. J. H. MASON KNOX, JR., PH.D., M.D., BALTIMORE 47

INTRAVENOUS GLUCOSE INJECTIONS IN INFANCY. CHARLES HUNTER DUNN, M.D., BOSTON 52

POSTOPERATIVE ACID INTOXICATION. P. C. JEANS, M.D., AND MEREDITH R. JOHNSTON, M.D., ST. LOUIS 57

INTUSSUSCEPTION OF THE BOWEL IN AN INFANT, FOLLOWED BY OBSTRUCTION OF THE BOWELS WITH A SECOND INTUSSUSCEPTION WITHIN THREE WEEKS AFTER THE FIRST OPERATION. A. PESKIND, M.D., CLEVELAND 63

PROGRESS IN PEDIATRICS:

CURRENT PEDIATRIC LITERATURE 65

AUGUST, 1917. NUMBER 2

PAGE

EPIDEMIOLOGIC DATA IN THE POLIOMYELITIS EPIDEMIC IN NEW YORK STATE. MATTHIAS NICOLL, JR., M.D., ALBANY, N. Y. 69

INFANT WELFARE WORK IN WAR TIME. GRACE L. MEIGS, M.D., WASHINGTON, D. C. 80

THE URIC ACID CONTENT OF THE BLOOD IN THE NEW-BORN. J. P. SEDGWICK, B.S., M.D., AND F. B. KINGSBURY, PH.D., MINNEAPOLIS 98

THE EFFECT ON HUMAN MILK PRODUCTION OF DIETS CONTAINING VARIOUS FORMS AND QUANTITIES OF PROTEIN. B. RAYMOND HOOBLER, M.D., DETROIT 105

CONGENITAL SKIN DEFECTS. ISAAC A. ABT, M.D., CHICAGO 113

PROGRESS IN PEDIATRICS:

REVIEW OF POLIOMYELITIS. A. B. SCHWARTZ, M.D., MILWAUKEE, WIS. 122

CURRENT PEDIATRIC LITERATURE 149

SEPTEMBER, 1917. NUMBER 3

PAGE

OBSERVATIONS ON THE PRESENCE OF THE BACILLUS ABORTUS BOVINUS IN CERTIFIED MILK. E. C. FLEISCHNER AND K. F. MEYER, SAN FRANCISCO 157

THE USE OF PANCREATIC VITAMIN IN CASES OF MARASMUS. WALTER H. EDDY, PH.D., AND JOSEPH C. ROPER, M.D., NEW YORK 189

VARIATIONS IN INFANTS OF TOTAL BLOOD SOLIDS AND THE CONCENTRATION OF SODIUM CHLORID IN THE PLASMA. ANGELIA M. COURTNEY AND HELEN L. FALES, NEW YORK 202

RUMINATION IN THE FIRST YEAR OF LIFE. C. G. GEULFE, CHICAGO 210

GLIOSARCOMA IN AN INFANT OF SEVEN WEEKS RESEMBLING HYDROCEPHALUS. L. EMMETT HOLT, M.D., LL.D., NEW YORK 219

THE EFFECT OF COD LIVER OIL ON GROWTH IN A CASE OF INTESTINAL INFANTILISM. L. EMMETT HOLT, M.D., LL.D., ANGELIA M. COURTNEY AND HELEN L. FALES, NEW YORK 222

PROGRESS IN PEDIATRICS:

INDEX OF CURRENT PEDIATRIC LITERATURE 228

American Journal of Diseases of Children

Vol. 14 DECEMBER, 1917 No. 6

THE DIAGNOSIS OF THE AGE OF THE FETUS BY THE USE OF ROENTGENOGRAMS *

JULIUS H. HESS, M.D.
CHICAGO

The determination of the age of the fetus is of considerable practical importance. On the age of the fetus and on its development are based the chief factors that are to be considered before giving prognosis as to the possibility of saving the life of the prematurely born infant. The method of treatment, and especially that of feeding, depends primarily on the age of such infant. In medicolegal cases the question of the age of the fetus may occasionally be of paramount importance, and when only portions of the fetus are available for examination, the roentgenographic method offers the best means for arriving at a diagnosis of the age of such fetus.

Time of Conception.—The age of the fetus is the time which has elapsed from conception; that is, from the time of the fertilization of the ovum from which the fetus developed. The time of conception cannot be accurately determined. Conception does not follow immediately after insemination, and its relation to menstruation is so uncertain that an error of one month is possible in attempts to calculate the age of the fetus from the time of menstruation. A number of observers, Arnold,[1] Bischoff,[2] Dalton,[3] Leopold and Mironoff,[4] Leopold and Ravano,[5] and Reichert[6] have found that menstruation and ovulation usually occur at the same time, but that ovulation may take place at

* Submitted for publication Sept. 15, 1917.

* From the Departments of Pediatrics and of Anatomy, University of Illinois, College of Medicine.

1. Arnold: Inaug. Dissert., Würzburg, 1887.

2. Bischoff: Beweis der von der Begattung unabhängigen periodischen Reifung und Lösung der Eier der Säugetiere und des Menschen als erste Bedingung ihrer Fortpflanzung. Giessen 1844. Entwickelung des Hundeeies, 1845.

3. Dalton: The Corpus Luteum of Menstruation and Pregnancy. Philadelphia, 1851.

4. Leopold and Mironoff: Arch. f. Gynäk., 1894, **14.**

5. Leopold and Ravano: Arch. f. Gynäk., 1907, **83.**

6. Reichert: Beschreibung einer frühzeitigen menschlichen Frucht im blasenförmigen Bildungszustande. Abhandl. d. k. preuss. Akad. d. Wissensch., Berl., 1873.

any time during the intermenstrual period and that, on the other hand, menstruation may occur without ovulation.

Issmer[7] found that when pregnancy was calculated from the first day of the last menstruation it averaged 280 days in 1,220 cases, while in 628 cases it averaged 269 when estimated from a fruitful copulation.

Mall,[8] in his studies on the determination of the age of human embryos and fetuses, comes to the conclusion that "the most probable time of conception is during the first week after the menstruation, as advocated by Hensen[9] and other obstetricians, and that in determining the age of human embryos it is probably more nearly correct to count from the end of the last period, for all evidence points to that time as the most probable at which pregnancy takes place."

His conclusions are also confirmed by recent observations of Siegel[10] on 100 pregnant women in whom the day of an isolated intercourse could be determined, this being made possible by the conditions during the present war. He concludes that conception can only take place during the first twenty-one days after the last period, and that the most susceptible time is before the sixth day. In no case could conception be established after the twenty-first day, so that he thinks it is safe to say that the postmenstrual period is the most favorable time for fertilization.

According to all this, therefore, conception in most cases occurs shortly after menstruation, and the age of the fetus is to be counted from the last day of menstruation. But even using this method of computing the age, there is room for an error in some cases which may amount to as much as one month.

Length of the Fetus.—If the date of the last menstruation or the date of a single isolated coitus is not known, then estimation of the age has usually been made on the basis of the length of the fetus, which has been generally regarded as the safest guide in the determination of the age.

The average lengths of normal fetuses as given by different authors are shown in Table 1.

Issmer[7] estimated the length of pregnancy and the age of the child in a number of cases and gives the figures in Table 2 as to the length and the age of the baby.

The same author (Issmer) has observed that in children of the same length differences of ten to eighteen days in pregnancy are possible.

---

7. Issmer: Ueber die Zeitdauer der menschlichen Schwangerschaft. Arch. f. Gynäk., 1887, **30,** 277 and 1889, **35,** 310.
8. Mall. Keibel-Mall: Manual of Human Embryology, **1,** 196.
9. Hensen: Hermann's Handbuch der Physiologie, 1881, **6.**
10. Siegel: Deutsch. med. Wchnschr., 1915, No. 2.

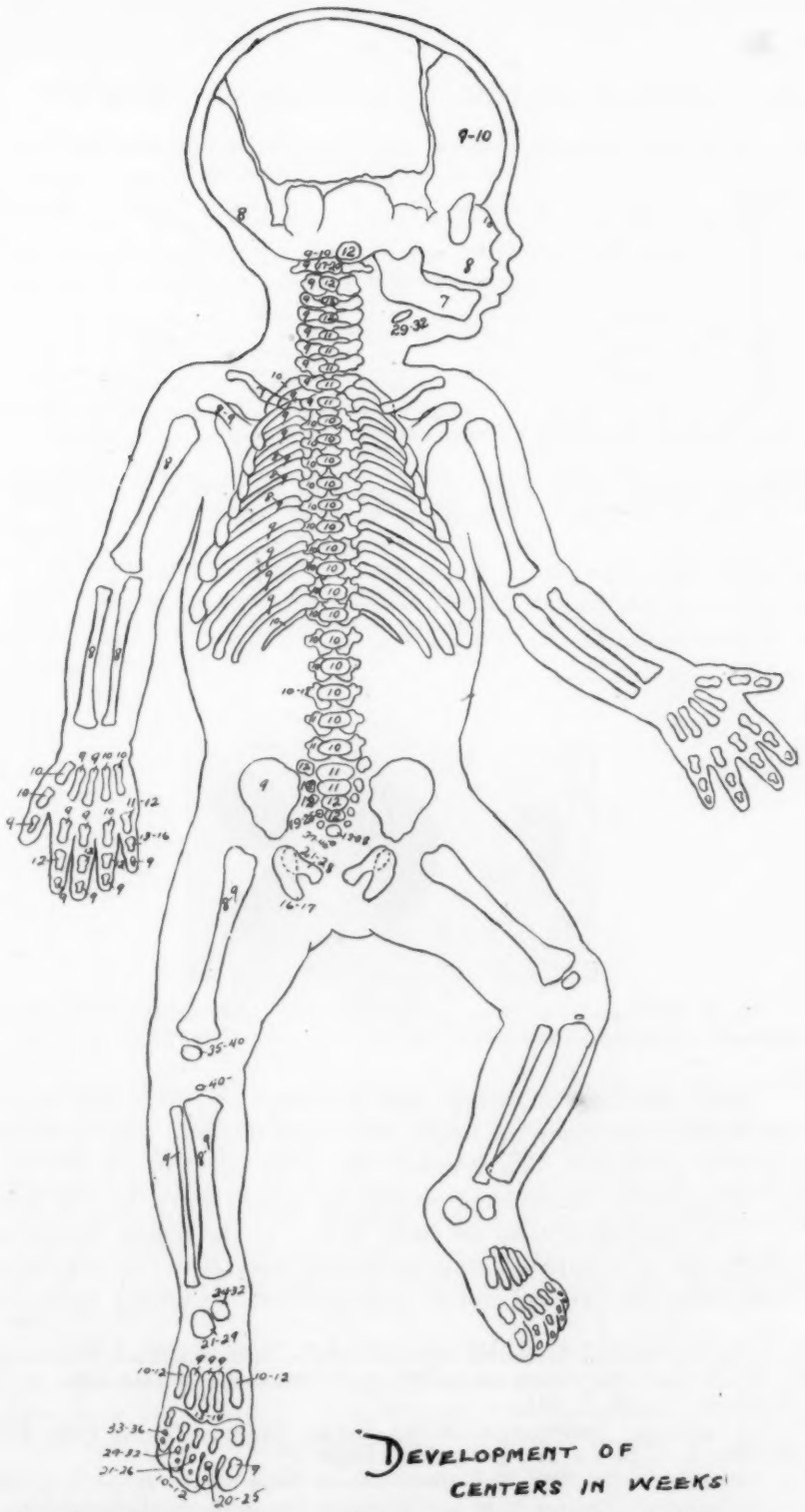

Fig. 1.—Diagram showing osseous development of infant at full term, and development of ossification centers in weeks. Centers shown which are frequently absent at birth: (1) head of tibia; (2) coccyx. Centers omitted in outline: (1) sternum; (2) hyoid.

TABLE 1.—AVERAGE LENGTHS IN CENTIMETERS OF NORMAL FETUSES AS GIVEN BY DIFFERENT OBSERVERS

Lunar Months	Mall[9]	Von Winckel[11]	De Lee[12]	Lambertz[13]	Ahlfeld[14]	Schroeder[15]
1st*	0.25	.....	0.75-0.9			
2d	0.55- 3.0	0.9-2.5	2.5			
3d	4.1 - 9.8	7-9	7-9	6-11		
4th	11.7-18.0	10-17	10-17	11-17		
5th	19.8-25.0	18-27	17-26	17-28		
6th	26.8-31.5	28-34	28-34	26-37		
7th	33.1-37.1	35-38	35-38	35-38	36-40	
8th	38.4-42.5	40-43	43	38-42	40-43	41.2
9th	43.6-47.0	46-48	46-48	42-45	46-48	44.6
10th	48.4-50	48-50	48-50	45-52	48-50	46.0

* The length for the first two months represents the measurement from the vertex to the buttocks; all the other measurements are from vertex to sole.

TABLE 2.—SIZE OF THE CHILD IN CENTIMETERS. NUMBER OF CASES AND AGE IN DAYS AS CALCULATED FROM THE BEGINNING OF THE LAST MENSTRUATION

Size	Number Cases	Age, Days
48	203	271
49	272	279
50	252	277
51	211	282
52	123	283
53	34	286
54	18	290

Fig. 2.—Photographs of fetus (exact size): *a*, at 4 to 5 weeks; *b*, 5 to 6 weeks, and *c*, 6 to 7 weeks, respectively.

There are many physiologic and pathologic variations and numerous factors influencing the length, the weight and also other measurements of the fetus. Von Winckel[11] gives the length of the new-born as 48 to 56 cm., but children over 60 cm. have frequently been born.

Some children at term are small because of the general debility of the mother or because of acute or chronic diseases during pregnancy. Diseases of the placenta and its abnormal location usually retard the

11. Von Winckel: Handbuch der Geburtshülfe, 1903, Bergmann, Wiesbaden.
12. De Lee: The Principles and Practice of Obstetrics. Philadelphia: W. B. Saunders Co., Ed. 2, 1915.
13. Lambertz: Development of the Human Skeleton During Fetal Life. Fortschr. a. d. Geb. d. Röntgenstrahlen, Suppl. 1.
14. Ahlfeld: Von Winckel's Handbuch der Geburtshülfe, **1**, No. 1, p. 290.
15. Schroder: Quoted from von Winckel's Handbuch der Geburtshülfe.

growth of the fetus. In successive pregnancies the children at term usually increase in size.

Other Measurements of the Fetus.—Von Winckel[11] regards the circumference of the head as of importance for the diagnosis of the age of the fetus and gives the following figures:

4th month.....10-14 cm.	8th month.....25-30 cm.
5th month.....13-18 cm.	9th month.....29-33 cm.
6th month.....19-24 cm.	10th month.....32-37 cm.
7th month.....23-28 cm.	

The weight is entirely unreliable for the estimation of the age of the fetus, because it is subject to too many variations and is much influenced by the mother's general condition, and more especially by her diet.

Thus, it is seen that even the length, which up to this time has been regarded as the most reliable criterion for the determination of the age of the fetus, has many shortcomings and allows of an error of several weeks.

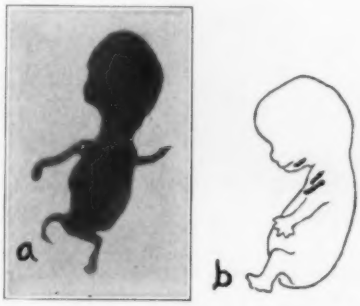

Fig. 3.—Roentgenogram (*a*) and diagram (*b*) of fetus at 7 weeks, actual size.

Roentgenograms of the Normal Development of the Skeleton.—Since roentgenograms have been found of much value in diagnosis of various diseases and conditions it was hoped that they might be of service in the diagnosis of the age of the fetus and that the development of the fetus as observed with the aid of roentgen rays might be a reliable guide for the determination of its age. The developing bones of the fetus with their centers of ossification are the only organs suitable for this purpose.

Basing our facts on the roentgenographic studies of a series of fifty-five normal cases collected during the past year and whose ages have been determined from the history of menstruation and of pregnancy and from their measurements, the normal process of development of the human skeleton as observed in roentgenograms is found to be as follows:

The ossification of the human skeleton begins in the upper part of the body and spreads very rapidly in both directions.

Seventh Week.—The first centers of ossification develop in the clavicles in the sixth to seventh week of intra-uterine life (Keibel-Mall,[8] Rauber-Kopsch,[16] but they do not become visible in the roentgenograms until in the seventh week. The ossification center appears in the middle of each clavicle and spreads rapidly in both directions.

Soon after the ossification has started in the clavicle, one center appears in each half of the mandible.

Outside of these centers of ossification usually no other centers, except occasionally that of the maxilla, are visible in roentgenograms of the 7 weeks' old fetus.

Eighth Week.—Osseous development makes rapid progress in the eighth week, and a large number of centers of ossification become visible at this time.

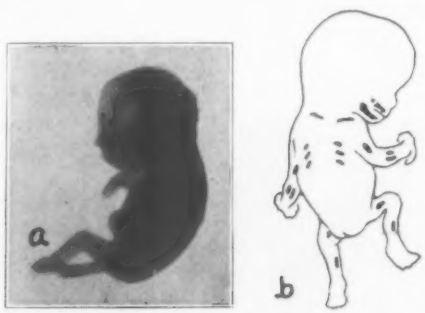

Fig. 4.—Roentgenogram (*a*) and diagram (*b*) of fetus at 8 weeks, actual size.

The following bones show centers of ossification demonstrable in roentgenograms:

Skeleton of the head: The squamous portion of the occipital bone and superior maxilla. In the latter the ossification begins soon after that of the mandible, the center appearing above the region where the alveolus of the incisor tooth is later located.

Usually no centers of ossification are present in the axial skeleton in this week.

Shoulder girdle: In the scapula a center of ossification usually appears in the eighth week, sometimes in the ninth week. The center corresponds to the position of the middle of the spine of the scapula.

Upper extremity: The humerus is the first bone of the free extrem-

16. Rauber-Kopsch: Lehrbuch der Anatomie des Menschen. Thieme, Leipsic, Ed. 10, 1914, **2**.

ities to show a center of ossification, which appears in the diaphysis early in the eighth week. Radius and ulna follow in the order given, the centers appearing very soon after those of the humerus.

The ribs start in their ossification in the eighth week, an ossification center appearing in the region of the angle and extending slowly toward the vertebral column, but rapidly in the opposite direction. The fifth, sixth and seventh ribs, which ossify first, are visible in this period. From this region the process of ossification progresses with equal rapidity both cephalad and caudad. The last rib to ossify is usually

TABLE 3.—TIME OF APPEARANCE OF CENTERS OF OSSIFICATION

HEAD

Mandible	7th week
Occipital Bone (squamous portion)	8th week
(lateral and basilar p.)	9th-10th week
Superior maxilla	8th week
Temporal bone (petrous, mastoid and zygoma)	9th week
Sphenoid (inner lamella of pterygoid process)	9th week
(great wings)	10th week
(lesser wings)	13th week
(anterior body)	13th-14th week
Nasal bone	10th week
Frontal bone	9th-10th week
Bony labyrinth	17th-20th week
Milk teeth (rudiments)	17th-28th week
Hyoid bone (greater cornua)	29th-32d week

TABLE 4.—TIME OF APPEARANCE OF CENTERS OF OSSIFICATION

SHOULDER GIRDLE

Clavicle (diaphysis)	7th week
Scapula	8th-9th week

UPPER EXTREMITY

Humerus (diaphysis)	8th week
Radius (diaphysis)	8th week
Ulna (diaphysis)	8th week
Phalanges, terminal	9th week
basal, 3d and 2d	9th week
basal, 4th and 1st	10th week
basal, 5th	11th-12th week
middle 3d, 4th, 2d	12th week
middle 5th	13th-16th week
Metacarpals, 2d and 3d	9th week
4th, 5th, 1st	10th-12th week

the first pair. Shortly before the first pair, the twelfth pair usually ossifies, but this is very irregular and we found it absent in several of our cases in old fetuses when other bones of the body and all the other ribs were very well developed.

Lower extremity: Centers of ossification may be occasionally seen in the diaphyses of the femur, but usually they become visible in the ninth week. The femur is the first to show a center, the tibia starting in its ossification a little later, and the fibula following very soon after the tibia.

Ninth Week.—Portions of the hand and of the foot enter the stage of ossification, these being the most important new developments in this week.

The following additional centers of ossification are visible in the head: inner lamella of the pterygoid process of sphenoid and mastoid portions of the temporal bone. The zygomatic process of the temporal bone begins to cast a shadow, its shape being somewhat pointed anteriorly and somewhat convex externally, thus resembling a needle. Bony trabeculae are often seen in the posterior root of the mastoid process. The superior maxilla forms at this time a simple triangular plate, the

TABLE 5.—TIME OF APPEARANCE OF CENTERS OF OSSIFICATION

AXIAL SKELETON (VERTEBRAE)

Arches, all cervical and upper 1 or 2 dorsal	9th week
all dorsal and 1 or 2 lumbar	10th week
lower lumbar	11th week
upper sacral	12th week
4th sacral	19th-25th week
Bodies from 2d dorsal to last lumbar	10th week
from lower cervical to upper sacral	11th week
from upper cervical to lower sacral	12th week
5th sacral	13th-28th week
1st coccygeal	37th-40th week
structural arrangement	13th-16th week
odontoid process of axis	17th-20th week
Costal processes, 6th and 7th cervical	21st-33d week
5th cervical	33d-36th week
4th, 3d, 2d cervical	37th-40th week
Transverse processes, cervical and dorsal	21st-24th week
lumbar	25th-28th week

RIBS AND STERNUM

Ribs, 5th, 6th, 7th	8th-9th week
2d, 3d, 4th, 8th, 9th, 10th, 11th	9th week
1st	10th week
12th (very irregular)	10th week

STERNUM

Sternum	21st-24th week

base of which is parallel to the margin of the maxilla, the apex pointing towards the root of the nose. The malar bone may become visible towards the end of this week or during the next week.

Axial skeleton: Arches of all the cervical and upper one or two dorsal vertebrae show centers of ossification, usually no centers for bodies being visible. One center develops in each arch, the process beginning in the first cervical vertebra and proceeding caudally.

Shoulder girdle: The acromion process of the scapula begins to ossify.

Upper extremity: The terminal phalanges of all fingers begin to ossify in this week. The first beginnings of these centers are difficult to study in roentgenograms on account of their small size, but the later

stages can be easily demonstrated. Development of the centers of ossification in terminal phalanges is followed by the appearance of centers in the metacarpals which become visible in the ninth to tenth week. The following is the order of ossification in the metacarpals: second, third, fourth, fifth, first, of which the second and the third are usually visible in the ninth week.

Ribs: All the ribs, except the first and the twelfth cast shadows.

Pelvic girdle: The ilium usually appears in this week, rarely at the end of the eighth week. Ossification begins in the region of the greater sacrosciatic foramen and near the acetabulum.

Lower extremity: Centers of ossification in femur, tibia and fibula are seen. Centers begin to develop in the phalanges, the first one being

TABLE 6.—TIME OF APPEARANCE OF CENTERS OF OSSIFICATION

PELVIC GIRDLE	
Ilium	9th week
Ischium (descending ramus)	16th-17th week
Os pubis (horizontal ramus)	21st-28th week
LOWER EXTREMITY	
Femur (diaphysis)	8th-9th week
(distal epiphysis)	35th-40th week
Tibia (diaphysis)	8th-9th week
(proximal epiphysis)	40th week
Fibula	9th week
Os calcis	21st-29th week
Astragalus	24th-32d week
Cuboid	40th week
Metatarsal, 2d and 3d	9th week
4th, 5th and 1st	10th-12th week
Phalanges, terminal 1st	9th week
terminal 2d, 3d, 4th	10th-12th week
terminal 5th	13th-14th week
basal 1st, 2d, 3d, 4th, 5th	13th-14th week
middle 2d	20th-25th week
middle 3d	21st-26th week
middle 4th	29th-32d week
middle 5th	33d-36th week

a center for the diaphysis of the terminal phalanx of the big toe. Diaphyses of the metatarsals follow in the same sequence and almost at the same time as corresponding portions of the hand, but with far less regularity.

Tenth Week.—Comparatively few new centers of ossification are added in this week.

Skeleton of the head: Nasal bone and frontal bone show centers of ossification. The great wing of the sphenoid becomes visible.

Axial skeleton: Bodies of the vertebrae begin to cast shadows. The process starts in the bodies of the lower dorsal vertebrae, progressing from this region with unequal rapidity in both directions. Usually the lower ten dorsal and all the lumbar vertebrae show centers of ossi-

fication in their bodies in this week. The process of ossification of the arches, progressing downward, has affected usually all the thoracic vertebrae, being more or less advanced occasionally, invading often the upper lumbar region.

Shoulder girdle: Ossification of the scapula spreads to the supraspinous fossa.

Upper extremity: Diaphyses of basal phalanges of fingers develop centers of ossification, the following being the sequence: third, second, fourth, first and fifth. Of these, usually the third, only, shows center in this week.

Ribs: At this time ossification, as a rule, is seen in all the ribs, the twelfth behaving very irregularly. It was found absent in some comparatively old fetuses far beyond the tenth week.

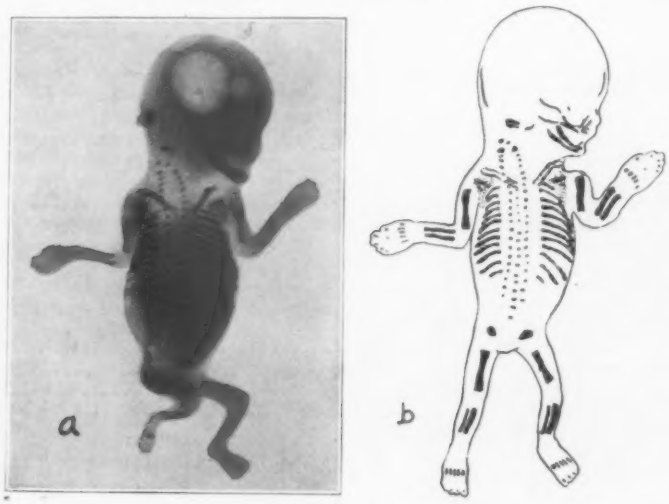

Fig. 5.—Roentgenogram (*a*) and diagram (*b*) of fetus at 10 weeks, actual size.

Lower extremity: Beginning with this week centers of ossification are present also in the terminal phalanges of the second and of the third toes.

Eleventh to Twelth Week.—In this period almost as many centers of ossification are present in the fetal skeleton as at the time of birth, so that but few are added during the period of development following the third month, and further changes in the fetal skeleton consist mostly of growth of the centers of ossification, of their fusion and of the formation of the internal structure of the bones. A fine, somewhat irregular, medullary cavity forms in the long bones, usually being seen first in the tibia.

Skeleton of the head: The tympanic ring usually becomes visible in this week, rarely at the end of the eleventh week. In pictures taken from the side, its shadow lies between the angle of the mandible and the basilar portion of the occipital bone. The median lamella of the pterygoid process reaches considerable size and is visible as a hook-shaped, curved plate with concavity posteriorly, lying behind the lower portion of the perpendicular part of the palate bone. The malar bone joins the end of the zygomatic process of the superior maxilla and that of the temporal bone. Four centers are now present in the occipital bone. The anterior sphenoidal body begins to ossify.

Axial skeleton: Ossification of the arches invades the lower lumbar region. The ossification of the bodies now appears in the lower cervical

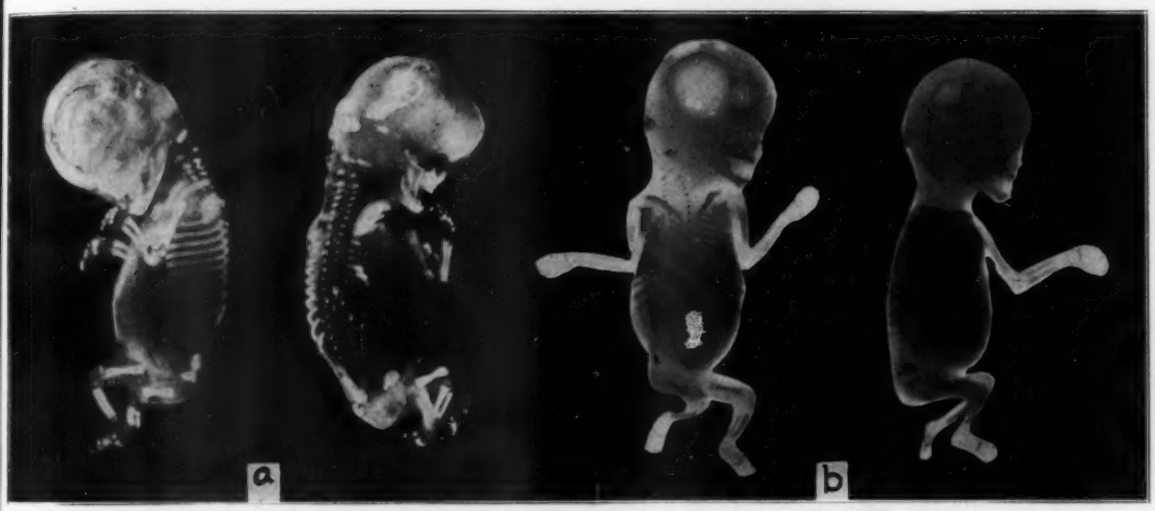

Fig. 6.—Photograph (*a*) and roentgenogram (*b*) of transparent specimens of fetus at 10 weeks, actual size.

region and in the upper part of the sacrum, the intermediary bodies having been visible previously. There are, however, considerable variations in the time of appearance of centers of ossification in the sacral vertebrae.

Shoulder girdle: No new centers develop, the old ones increasing in size.

Upper extremity: The diaphyses of all the basal phalanges cast shadows. Middle phalanges of the third, fourth and occasionally of the second finger develop centers of ossification in their diaphyses. The middle phalanx of the fifth finger ossifies much later. Up to the end of the third month the bony diaphyses of the humerus, radius and

ulna remain longer and thicker than the corresponding bones of the lower extremity.

Pelvic girdle: Either in this period or shortly after, a third center of ossification develops in the ilium, being situated ventrally from the fused first and second centers. There is a marked irregularity in the time of appearance of the third center of the ilium, since occasionally it may appear almost three weeks after this time.

Lower extremity: The terminal phalanges of the fourth and fifth toes usually develop centers of ossification; in the fifth, however, the

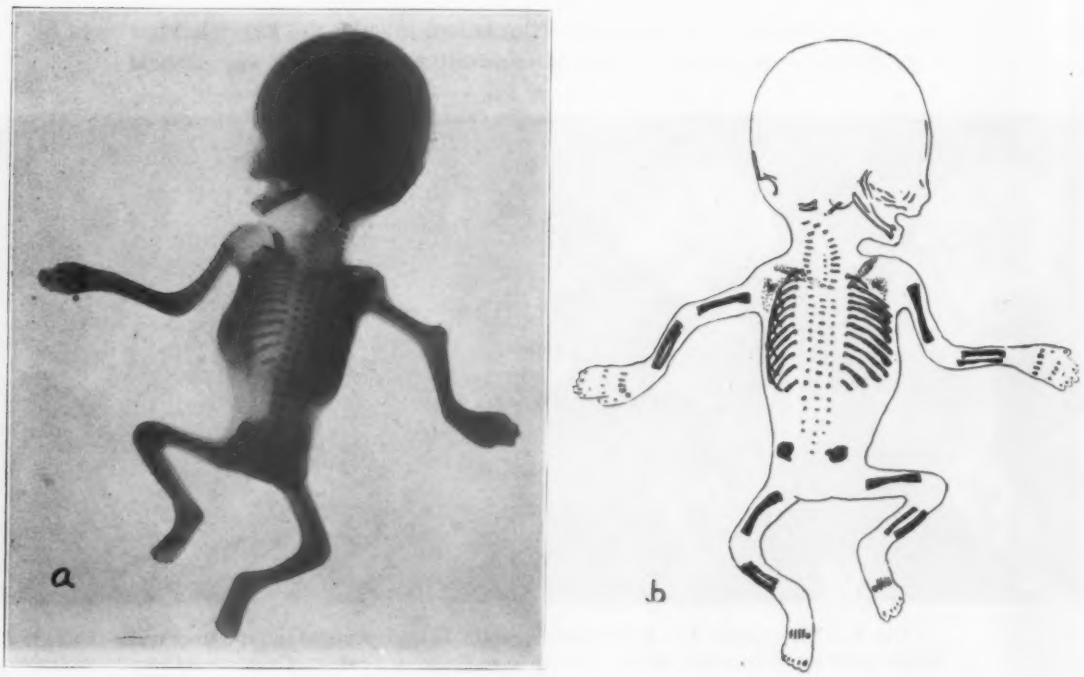

Fig. 7.—Roentgenogram (*a*) and diagram (*b*) of fetus at 11 to 12 weeks, actual size.

center may occasionally appear as late as in the thirteenth week. The bony diaphysis of the femur, which up to this time has been shorter and thinner than the bony diaphysis of the humerus, has almost reached the length of the latter, remaining, however, still somewhat thinner.

Thirteenth to Sixteenth Week.—Characteristic in the osseous development of this period is the appearance of structural arrangement in the bodies of some vertebrae and the presence of centers of ossification in the diaphyses of all of the long bones of the hand and of the foot, except the middle phalanges of toes.

Skeleton of the head: The lesser wing of the sphenoid is visible at the beginning of this period. The posterior body of the sphenoid appears about the fourteenth week.

Axial skeleton: At the end of this period all the vertebrae, with the exception of first and second lower sacral and the coccygeal, have at least one center of ossification. Arches are ossified also in the upper sacral region and the bodies from the upper cervical down to the lower sacral region. Structural arrangement becomes visible in the bodies of some vertebrae. Upper and lower plate, casting denser shadow,

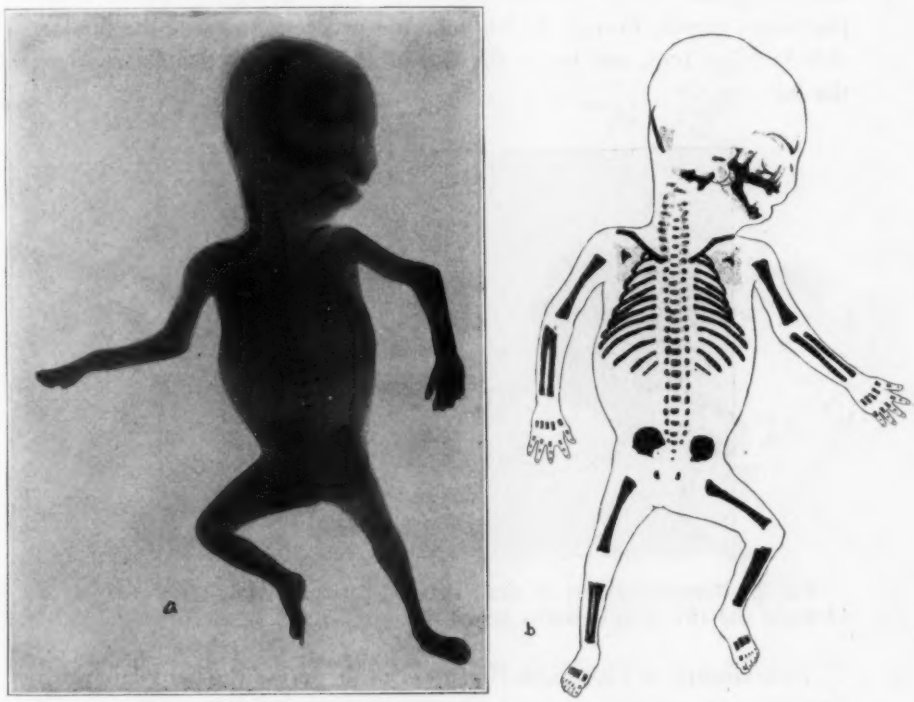

Fig. 8.—Roentgenogram (*a*) and diagram (*b*) of fetus at 13 to 16 weeks, one-half actual size.

become differentiated. A zone of lighter shadow is seen between these two plates and in the central portion of the body a flat, darker shadow appears. The greatest diameter of this darker shadow corresponds to the longitudinal axis of the fetus in lumbar and lower dorsal vertebrae; in other dorsal vertebrae it lies horizontally. These shadows appear in the bodies of the vertebrae in the region in which the primary centers made their first appearance.

Upper extremity: In the fifteenth to sixteenth week a center of ossification appears in the diaphysis of the middle phalanx of the fifth

finger, so that at this time the diaphyses of all the long bones of the hand possess centers of ossification.

Pelvic girdle: At the end of this period or somewhat later a center becomes visible in the descending ramus of the ischium. Instead of one center, two separate centers may develop in this portion of the innominate bone and they may remain separate for a long time afterwards.

Lower extremity: In the thirteenth week a center of ossification develops in the diaphysis of the terminal phalanx of the fifth toe, if it did not appear earlier. In the fourteenth week ossification in the basal phalanges begins, first in the big toe, and proceeds towards the fibular side in other toes, and up to the end of this period it usually reaches the last toe.

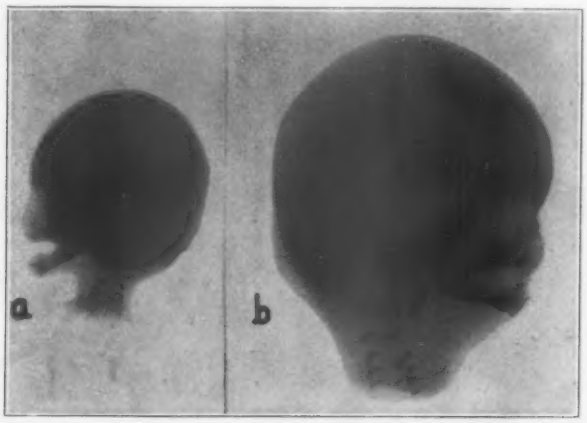

Fig. 9.—Roentgenograms of skull showing ossification centers at (*a*) 11 to 12 weeks and (*b*) 13 to 16 weeks, actual size.

Seventeenth to Twentieth Week.—In this period the bony labyrinth first appears and bone tissue begins to be formed in the rudiments of the milk teeth.

Skeleton of the head: Several new centers of ossification appear in the petrous portion of the temporal bone, but they do not show well in roentgenograms. The bony labyrinth starts in its development. In the rudiments of milk teeth, bone tissue begins to be formed and casts a shadow. The process starts in the lower incisors.

Axial skeleton: A center of ossification appears in the odontoid process of the axis. The darker shadows in the bodies of the vertebrae become more distinct and external formation and internal structure of osseous bodies of vertebrae become visible in roentgenograms. Ossi-

fication of the arches may reach the fourth sacral vertebrae at the end of this period, although this frequently occurs later.

Pelvic girdle: The twentieth week is the earliest time of appearance of a center in the horizontal ramus of the pubic bone; this, however, varies between the twentieth and the twenty-eighth week. The center is located near the margin of the obturator foramen, two centers developing occasionally.

Lower extremity: In the twentieth week a center of ossification may develop in the middle phalanx of the second toe, but this usually occurs in the twenty-first to the twenty-fourth week and frequently

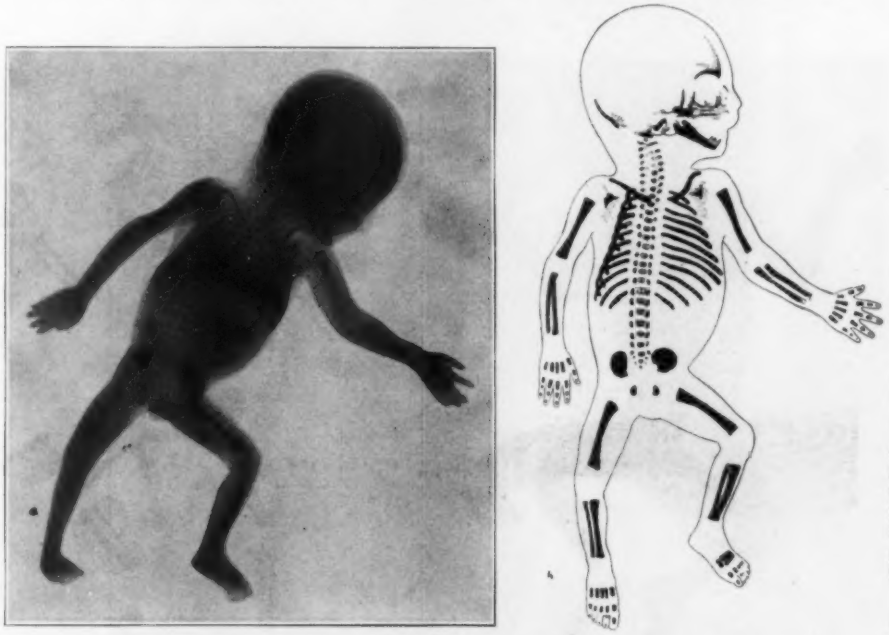

Fig. 10.—Roentgenogram (*a*) and diagram (*b*) of fetus at 17 to 20 weeks, one-third actual size.

even later than this. On the whole, there are marked differences and also individual variations in the time of appearance of centers of ossification, and also in the sequence of ossification in the phalanges of toes, especially in the basal phalanges and even more so in the middle phalanges. In the hand, however, the sequence of ossification in the phalanges is far more constant and the time of appearance of the centers is much less changeable than that of the centers in the phalanges of toes.

Twenty-First to Twenty-Fourth Week.—In this period ossification usually starts in the tarsus, os calcis being the first to show a center of

ossification. The sternum begins to develop by several centers of ossification, but there are considerable variations in the arrangement and size of these centers and also in the time of their appearance.

Skeleton of the head: The superior maxilla shows a large amount of spongiosa. Toward the twenty-fourth week the alveolar portion of the superior maxilla begins to overhang the level of the palatal plate, but develops as a real process only during the cutting of the teeth.

Axial skeleton: The costal process of the sixth cervical vertebra starts in its ossification. Shadows of transverse processes are seen in vertebrae down to the twelfth dorsal.

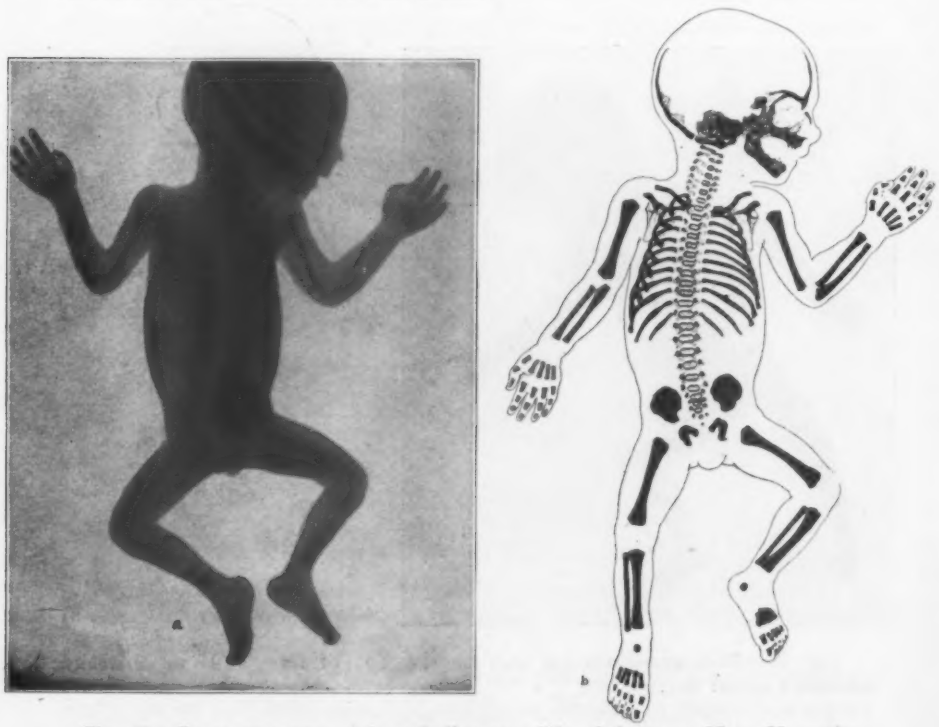

Fig. 11.—Roentgenogram (*a*) and diagram (*b*) of fetus at 25 to 28 weeks, one-fourth actual size.

Upper extremity: In this period the ossified portion of the diaphysis of the humerus reaches the articular ends and begins to overlap these so that at the distal end of the humerus both fossae (olecranon and cubital) and ulna and olecranon become visible, and later, on the proximal end of the humerus an indication of the medial and posterior portion of the neck appears.

The sternum starts in its ossification. Usually one center forms in the manubrium first and this is followed soon afterward by several

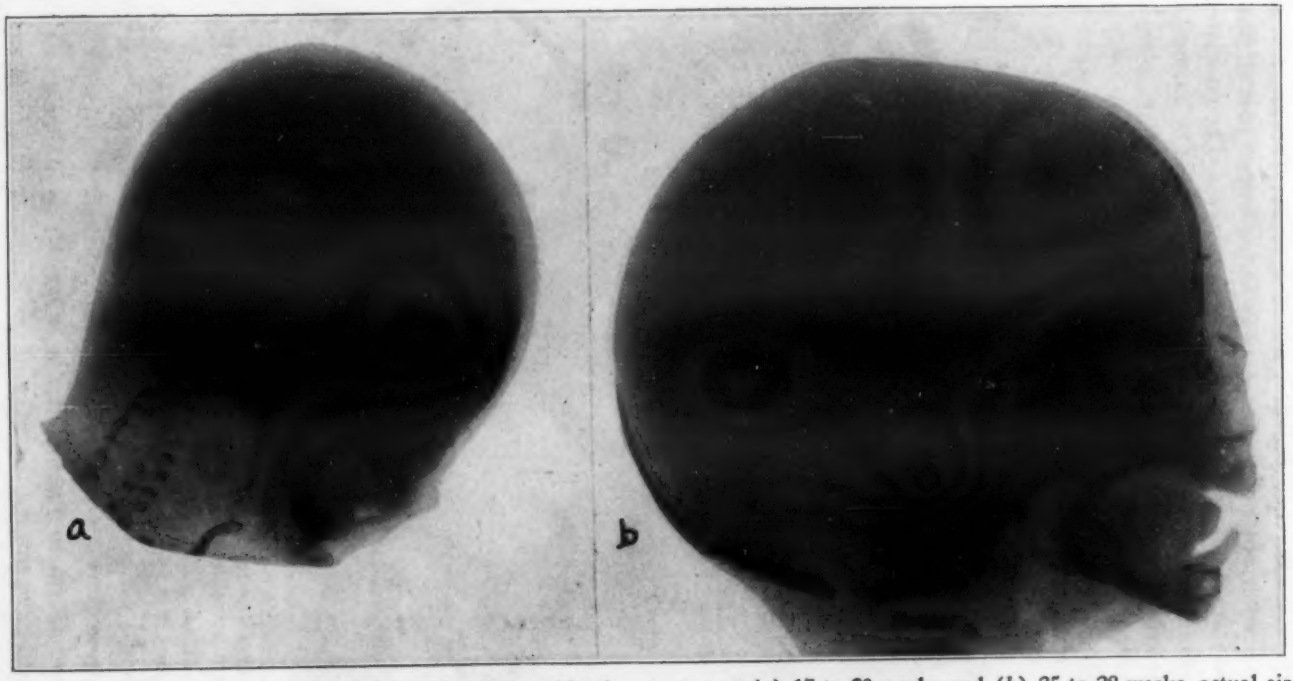

Fig. 12.—Roentgenograms of skull of fetus showing ossification centers at (*a*) 17 to 20 weeks and (*b*) 25 to 28 weeks, actual size.

centers in the body of the sternum. The centers form a longitudinal row first, and soon they assume a round or elliptical form. Not seldom the first centers of ossification appear in the upper part of the body between the second and the third costal cartilages. The position of the ossification centers of the sternum corresponds usually to the level of the intercostal spaces.

Lower extremity: A center of ossification develops in os calcis, its appearance being occasionally delayed by from four to eight weeks.

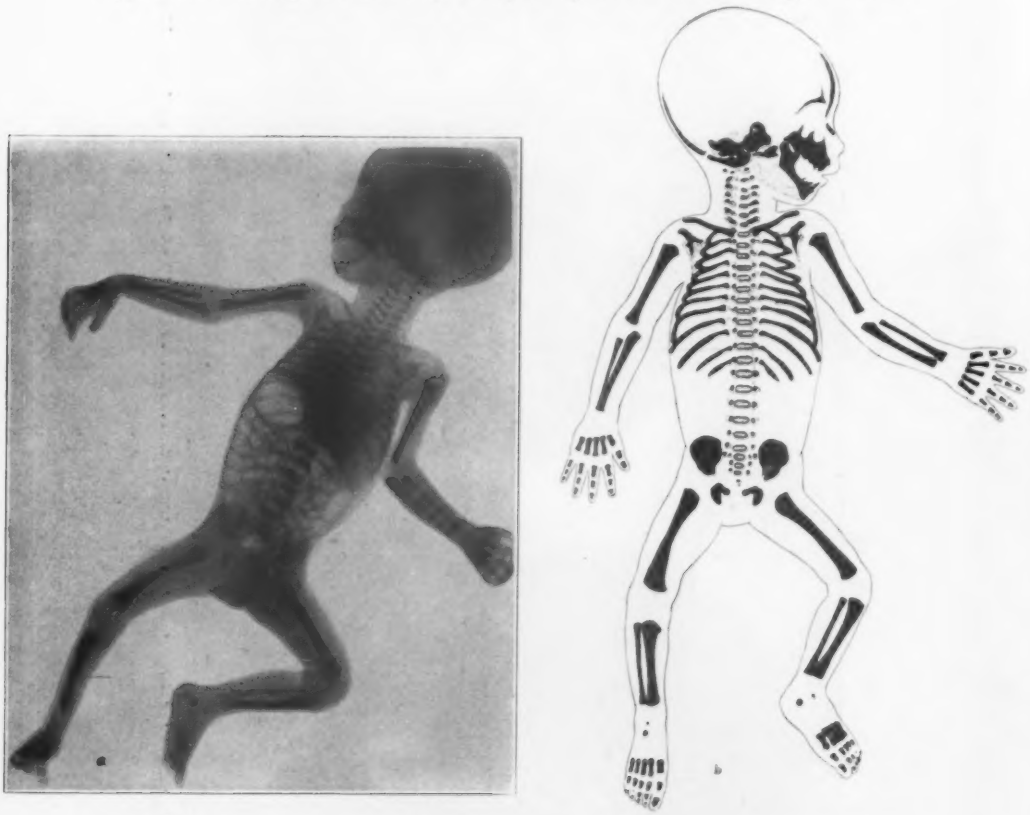

Fig. 13.—Roentgenogram (*a*) and diagram (*b*) of fetus at 29 to 32 weeks, one-fourth actual size.

Sometimes it is followed by the appearance of a center in the astragalus. The middle phalanx of the second toe, and occasionally that of the third toe, acquire a center of ossification in their diaphyses.

Twenty-Fifth to Twenty-Eighth Week.—The rudiments of all the milk teeth have entered the stage of ossification in this month.

The development of the transverse processes of the vertebrae progresses down to the last lumbar vertebra. At the end of this period a

center of ossification may develop in the lateral masses of the first and of the second sacral vertebrae. The body of the fifth and the arches of the fourth sacral vertebrae become ossified at this time, rarely earlier.

A center of ossification develops in the astragalus.

In the horizontal ramus of the pubic bone the center may develop as late as in this period.

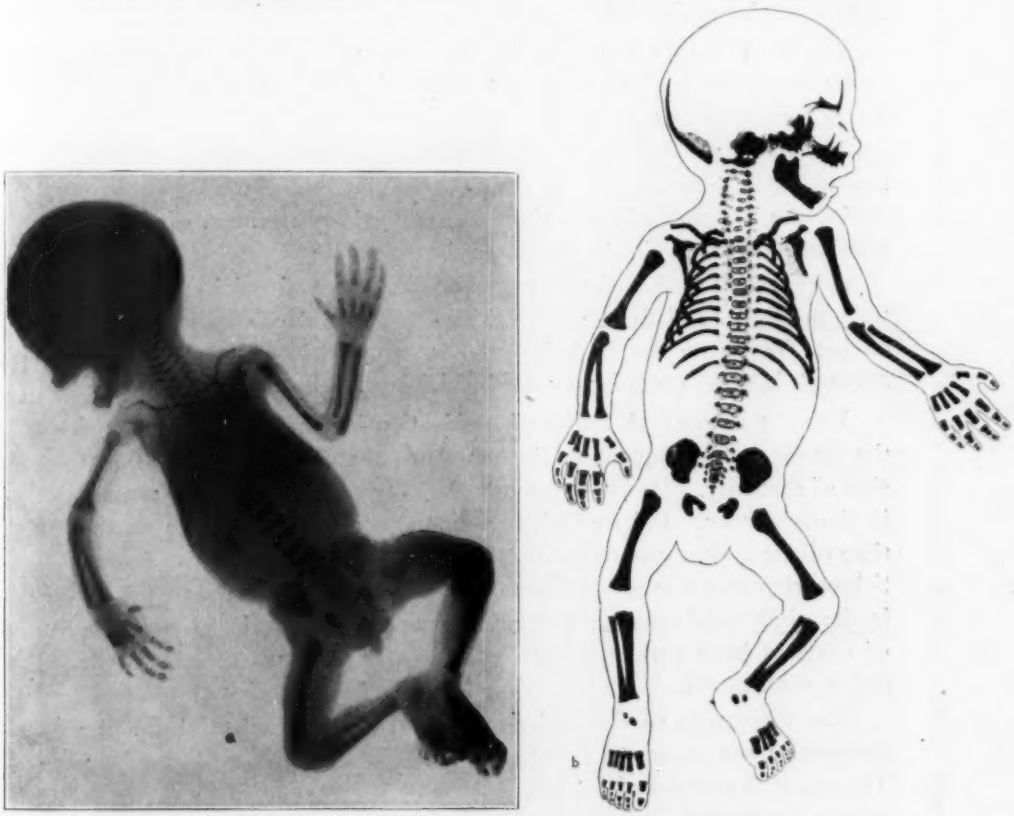

Fig. 14.—Roentgenogram (*a*) and diagram (*b*) of fetus at 33 to 36 weeks, one-fourth actual size.

Twenty-Ninth to Thirty-Second Week.—The greater cornua of the hyoid bone usually become visible, appearing as cone-shaped processes directed obliquely upwards at the level of the second cervical vertebra.

The lateral masses of the first and second sacral vertebrae ossify usually at this time.

In the sternum three or more large centers of ossification are visible.

The middle phalanx of the fourth toe frequently starts in its ossification during the period.

Thirty-Third to Thirty-Sixth Week.—This period is the earliest time at which the first epiphyseal center may appear, that of the distal epiphysis of the femur. Usually, however, this center appears later, at about the time of birth.

The costal process of the sixth and of the fifth cervical vertebrae start in their ossification.

Thirty-Seventh to Fortieth Week.—The middle turbinates ossify at the end of the fetal period and shortly before birth the rudiments of the first permanent molar teeth begin to ossify.

The costal process begins to ossify in the fourth, the third and the second cervical vertebrae; the first coccygeal vertebra usually ossifies during the last weeks before birth and vertical arrangement of trabeculae becomes visible in the bodies of the vertebrae.

A center of ossification appears in the proximal epiphysis of the tibia just before birth in a majority of cases, and ossification in the cuboid frequently starts before birth, usually by several centers, although in some cases it may not be visible even in the new-born.

The New-Born.—A center of ossification in the distal epiphysis of the femur is so frequent in the new-born that Lambertz[16] calls it a sign of maturity. This is frequently the only epiphyseal center present in the new-born. Poirier[17] gives a summary of the literature on the time of the appearance of the epiphysis at the distal end of the femur. Schwegel found it to appear between birth and the third year. Casper in the ninth fetal month. Hartmann found it lacking in 12 per cent. of cases at birth and in 7 per cent. of cases present as early as the eighth fetal month.

The four parts of the occipital bone (basilar, two lateral and the squamous) are separated from each other by thin layers of cartilage. The mastoid portion of the temporal bone is not ossified in its entire extent, a serrated line marking the boundary between bony and cartilaginous portions of the mastoid part. The lateral halves of the frontal bone are separated. The body of the hyoid bone is usually ossified. Both halves of the mandible, as a rule, are united by connective tissue.

The vertebrae are ossified in all their essential parts, including transverse and articular processes of the arches, but the centers of ossification are separated from each other by cartilage. The first coccygeal vertebra is usually ossified by this time.

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17. Poirier: Traite d'anatomie, **1**, 227.

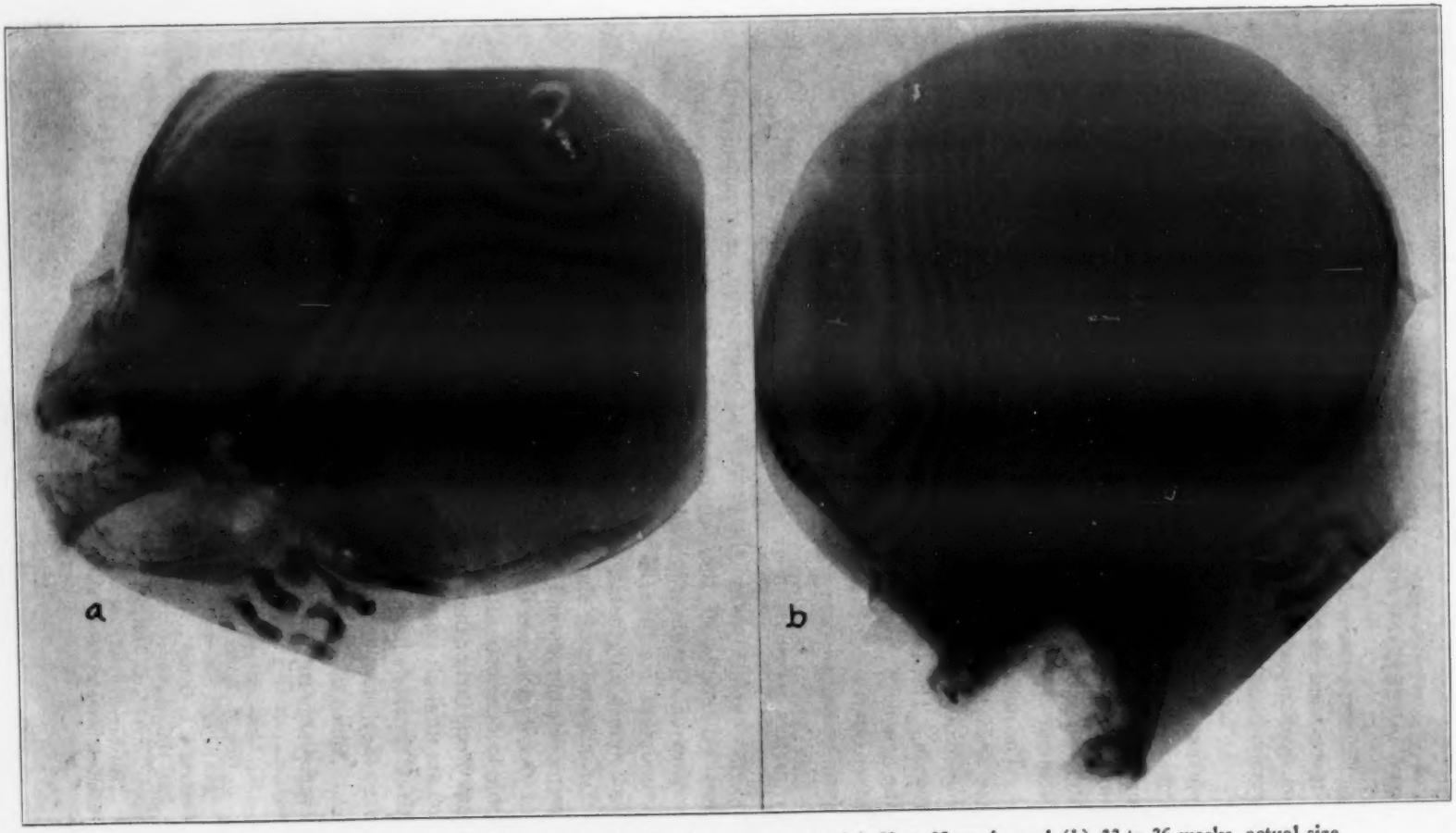

Fig. 15.—Roentgenograms of skull of fetus showing ossification centers at (*a*) 29 to 32 weeks and (*b*) 33 to 36 weeks, actual size.

In some cases the proximal epiphysis of the humerus is ossified. In the hand all bones are ossified except the carpus, in which centers of ossification in os magnum and unciform may be seen only very rarely.

At birth the ossified portion of the os pubis surrounds usually a portion of the anterior boundary of the obturator foramen, but the region of the symphysis and upper margin of the horizontal ramus of os pubis remain cartilaginous. The following portions of the innominate bone are not ossified in the new-born: the crest of the ilium with superior spines, acetabulum, spine of ischium and ascending ramus of ischium.

The middle phalanx of the fourth toe is frequently, that of the fifth toe always, cartilaginous in the new-born; in the fourth toe, however, the middle phalanx may start in its ossification in the eighth fetal month. The following portions of the leg are usually not ossified in the new-born: proximal epiphysis of tibia and of the fibula, epiphyses of metatarsal bones and of phalanges, the cuboid and the three cuneiform bones.

OTHER METHODS OF STUDYING OSSEOUS DEVELOPMENT COMPARED

We have compared the process of ossification, as observed in the roentgenograms of the fetuses studied with the roentgenographic studies of Alexander,[18] Bade,[19] Hasselwander[20] and Lambertz,[13] and found that the time of appearance of centers of ossification pretty well agrees, in general, there being minor differences only.

Compared with the studies of Mall, who used transparent specimens of embryos and fetuses for observing the appearance of centers of ossification, we find that by using transparent specimens he was able to demonstrate the minute centers of ossification generally about one week earlier than they are demonstrable by roentgenograms. This observation also agrees with textbooks of anatomy (Rauber-Kopsch, Gray[21]) which have been consulted for this purpose, and it is found that they place the time of appearance of various centers about one week ahead of the time at which the centers cast shadows in roentgenograms large enough to be visible.

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18. Alexander: The Development of the Osseous Vertebral Column. Fortschr. a. d. Geb. d. Röntgenstrahlen, Suppl. 13.

19. Bade: Short Description of Ten Roentgenologically Examined Fetuses. Centralbl. f. Gynäk., 1899, p. 1031.

20. Hasselwander: Studies of Ossification of the Skeleton of the Human Foot. Ztschr. f. Morphol. u. Anthropol., 1903, **5**, 438.

21. Gray-Spitzka: Anatomy Descriptive and Applied. Ed. 18. Lea & Febiger, Philadelphia and New York, 1910.

By courtesy of Dr. Roy Lee Moodie of the Department of Anatomy of the University of Illinois we obtained transparent specimens of a pair of twins from his embryologic collection and made roentgenograms of them. By studying these roentgenograms and specimens we found the following differences:

	Roentgenograms	Transparent Specimens
Basal phalanges of fingers	3d	2d, 3d, 4th
Terminal phalanges of toes	1st, 2d 3d	1st, 2d, 3d, 4th
Bodies of vertebrae	9 lower dorsal	9 to 10 lower dorsal, respectively
	All lumbar	All lumbar
	1st sacral	1st, 2d sacral
Arches of vertebrae	Upper 3 lumbar	Upper 3 to all lumbar, respectively

Thus the transparent specimens show in the tenth week centers that become visible in the roentgenogram only in the eleventh to twelfth week.

VARIATIONS IN OSSEOUS DEVELOPMENT

There are, as might be expected, some variations in the normal process of ossification, and it is also influenced by pathologic conditions of the mother and of the fetus (for example, syphilis, rickets, osteogenesis imperfecta, etc.). In general, these pathologic processes may well be diagnosed in the roentgenograms so that an error may easily be prevented. In some portions of the skeleton the ossification is less regular than in others, and as a general rule the more caudad the portions of the skeleton are, the more they are subject to variations in the process of ossification; and the centers which develop at a later period of fetal life are also more variable. Thus, there are considerable variations in the time of appearance of centers of ossification in the sacral vertebrae. The foot, as a general rule, is unreliable as an indicator of the age of the fetus. The ossification of the sternum is also irregular in the time of appearance, size and arrangement of the centers of ossification. The twelfth rib is also very irregular, and we found it absent in roentgenograms of the fetus from the thirteenth to sixteenth week, and also in some other older ones, although, as a rule, the twelfth rib appears in the tenth or in the eleventh week. Some of the centers, although demonstrable by careful examination, are so small as to be easily overlooked, and this may lead to an error. For this reason it is necessary to know what centers we may expect at that particular age of the fetus, and we should look for them in good light with a magnifying glass.

Bade[19] has examined roentgenograms of twin fetuses, one of which was 5.8 cm. long, weighing 8 gm., and the other 6.3 cm. long, weighing 11 gm. The only difference in the stage of ossification was that the

larger fetus showed two more centers in the arches of the vertebrae and two additional centers in terminal phalanges of the fingers.

In a fetus 7.1 cm. long and weighing 25 gm., the same author found, however, that the ossification did not progress as far as in the above mentioned twins, which were shorter. This delay in ossification was in the ribs (the last one not being visible, although all visible in the twins), while the axial skeleton showed only 5 bodies and 21 arches, as compared with 18 bodies and 22 and 24 arches, respectively, in the twins.

Alexander[18] found that while the ossification has been normal in other parts of the body, it has been occasionally delayed in the vertebral column.

He observed also twin fetuses measuring 8.9 cm. and 9.1 cm. in length, respectively. The only difference in ossification in these two fetuses was that in the larger one there was an ossification center in the third sacral vertebra, while in the shorter one the second sacral vertebra was the last one containing a demonstrable center.

In the twins from Dr. Moodie's collection which we have studied, the only differences in the stage of ossification are in the axial skeleton, one fetus showing centers for seventeen bodies and twenty-four arches on each side and the other only fifteen bodies and twenty-two arches on each side.

But in spite of these variations, the process of ossification seems to be more constant for a particular age than the length of the fetus. Mall,[22] in his article on ossification in embryos up to 100 days old, concludes that "the remarkable regularity of the appearance of the bones makes of them the best index of the size and of the age of embryo we now possess."

LIMITATIONS OF ACCURACY

In the first half of pregnancy the estimation of the age of the fetus may be made with greater accuracy because many more new centers appear in the first months, and also because the time of appearance of the earlier centers is more constant. In later months centers of the lower part of the skeleton mostly are available for study, and these are less constant in the time of their appearance. We have intentionally made our groupings broad enough to cover minor errors in diagnosis, but more careful subsequent studies may refine the diagnosis to such a degree that determination of age will be possible within the period of one week in the first half of the pregnancy, and within two weeks in the second half of the pregnancy.

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22. Mall: On Ossification Centers in Human Embryos Less Than One Hundred Days Old. Am. Jour. Anat., 1906, **5**, 433.

DIFFERENT VALUES OF THE DIFFERENT PORTIONS OF THE BODY

In the very early period (second month) the stage of ossification of clavicle and mandible is of chief importance, and on the basis of presence or absence of these centers determination of the age is made. Both roentgenograms and transparent specimens show that the time of appearance of these centers is almost constant, which makes them of cardinal value in diagnosis.

Next in importance are the centers of the upper extremity, and especially of the hand (metacarpals and phalanges) which are very regular, not only in the time of their appearance, but also in their sequence. The ossification of the diaphysis of the long bones of the arms extends from the eighth to the sixteenth week, and during this period the determination of the age may frequently be made from a good roentgenogram of the hand alone.

The progress of ossification of the head is also of considerable diagnostic importance, but the centers in many bones of the head are very difficult of demonstration. Those, however, that can be well demonstrated are of much value in the determination of the age. This is especially true of the occipital bone, superior maxilla, tympanic ring, nasal bone and hyoid bone.

The axial skeleton (the vertebral column) is less reliable than the foregoing named portions of the skeleton, and especially its lower portion is of little value in diagnosis of age. It is not the absolute number of arches or of the bodies ossified which decides the diagnosis as to the age of the fetus, but more the region involved and the extent of the development in the particular region of the vertebral column (cervical, dorsal, lumbar, sacral). On the other hand, however, the facts that the process of ossification of the vertebral column extends from the ninth week throughout the life of the fetus, and all its centers, as a rule, are well demonstrable, make it of especial value for at least approximate determination, although it must not be forgotten that occasionally the process of ossification may be delayed in the vertebral column, while it is normal and regular in other portions of the body.

The sternum is unreliable as an index of age and its centers are frequently difficult to demonstrate. The ribs are fairly constant, except the twelfth pair, which, as previously mentioned, may not show at all in roentgenograms of comparatively old fetuses.

While the ossifications of the long bones of the legs are pretty regular, since they appear at an early period, ossification in the foot is very irregular and the stage of ossification of the foot is of little

value in the determination of the age of the fetus. The osseous development of the foot extends from the ninth week to the end of the fetal period (not being, however, completed even at this time) and during this time there are very marked variations, especially in the centers which appear late in the fetal period.

From the above it may be seen that, as a general rule, the earlier a center appears the more regular it is, and since the process of ossification starts in the cephalic region and spreads caudally, it is also true that the more caudad a skeletal segment is situated the more it is subject to variations and irregularities.

ADVANTAGES OF THE ROENTGENOGRAPHIC METHOD

The peculiar advantage of the roentgenographic method for determining the age of the fetus lies in the fact that while in the determination of age according to the length we base our final conclusion usually on one, rarely on two or three measurements expressing different lengths of the fetus, in the roentgenographic method many centers of ossification are the factors taken into consideration before arriving at a final conclusion; and they act as check on each other and quite frequently the roentgenograms alone give us information as to whether the fetus is normal or not, a point which seldom may be determined from measurements alone.

TECHNIC

In studying the roentgenograms it is well to use a reading glass of about 4 inches in diameter, since some centers of ossification may be so small as to be very easily overlooked when sought for with the naked eye.

If only one exposure of the fetus is made, then the best position to show as many ossification centers as possible is as follows: The back lying flat on the plate, head turned completely to one side so that the side of the head lies on the plate and lateral exposure is obtained. (It should be remembered in the study of the skull that both halves of the skull are usually visible.) Arms and fingers should be extended and fingers spread as far as possible from one another. One hand should be pronated and the other supinated, the lateral exposure, which is often of so much value in roentgenograms taken for the purpose of surgical diagnosis, not being of much value, since in this position shadows of phalanges of fingers and of metacarpals are superimposed and cannot be well differentiated. The legs should also be extended and feet put into such a position that all metatarsals and phalanges are shown.

CONCLUSIONS

1. The stage of ossification of the skeleton of the fetus as observed in roentgenograms is of considerable practical importance in determining the age of the fetus.

2. The roentgenographic method of determining the age of the fetus is more reliable than determination of age based on length and other measurements, since osseous development is more regular and offers many more factors for consideration; one part of the body is a criterion of normality for the other parts, and pathology may often be readily recognized.

3. In the early months more accurate determination is possible than in the later months, and the roentgenographic method is undoubtedly capable of much greater refinement by further studies and observations.

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INTERNAL HYDROCEPHALUS

SECOND PAPER *

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In a recent communication a series of cases of internal hydrocephalus was presented and subdivided into two apparently distinct anatomic varieties — obstructive hydrocephalus and communicating hydrocephalus. These two groups were sharply differentiated by an anatomic difference, demonstrated by the introduction of a neutral solution of phenolsulphonephthalein into the cerebral ventricles and almost immediately testing for its presence in the spinal fluid. In the obstructive type, this solution introduced into the ventricles failed to appear in the spinal fluid; in the communicating type it appeared promptly in the spinal fluid.

Clinically, the two varieties appeared identical, and it was only by this test that hydrocephalus could be subdivided. The growth of the head seemed about equally rapid, the etiology was equally obscure, and either variety might be congenital or acquired.

By an estimation of the amount of phenolsulphonephthalein excreted by the kidneys following the intraventricular and intraspinal introduction, a quantitative absorption of cerebrospinal fluid could be estimated in each type of hydrocephalus and a second very important physiologic difference was demonstrated. In the obstructive type of hydrocephalus the absorption, which is restricted to the cerebral ventricles, is practically nil — less than 1 per cent., as contrasted with a normal of 12 to 20 per cent. (Fig. 2). In the communicating type, the absorption is 2 to 5 per cent., as contrasted with the same normal (Fig. 1). The similarity of the two groups is that in both there is a tremendous diminution in the absorption of cerebrospinal fluid. This is the reason hydrocephalus develops in both the obstructive and communicating types of hydrocephalus; that is, diminished absorption of cerebrospinal fluid with an unaffected production.

It was also shown by a series of experiments that cerebrospinal fluid is produced in the ventricles and is absorbed in the subarachnoid

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* From the Departments of Surgery and Pediatrics of the Johns Hopkins University and Hospital.

* First Paper: Dandy, W. E., and Blackfan, K. D.: Internal Hydrocephalus. An Experimental, Clinical and Pathological Study. Am. Jour. Dis. Child., 1914, **8**, 406. Also, Beitr. z. klin. Chir., 1914, **93**, 392. A preliminary report appeared in Jour. Am. Med. Assn., 1913, **61**, 2216.

space, and that the blood vessels of the entire spinal and cranial subarachnoid space participate in the absorption. It was further shown that in the obstructive type of hydrocephalus, the absorption of cerebrospinal fluid from the spinal subarachnoid space was normal. Hydrocephalus then resulted because a mechanical obstruction prevented the cerebrospinal fluid from leaving the ventricles of the brain, where there is no absorption, to the subarachnoid space, where the absorption may be normal.

In the communicating type of hydrocephalus there was a great diminution in the absorption of cerebrospinal fluid from the subarachnoid space. Although the cerebrospinal fluid passed freely from the ventricles, hydrocephalus resulted because an adequate absorption did not occur from the subarachnoid space.

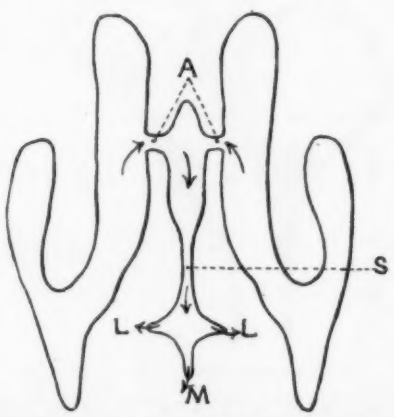

Fig. 1.—Diagram of the cerebral ventricular system, showing the intracerebral course of the cerebrospinal fluid. The three arrows at the base (*L*, *L*, and *M*) show the only three points of exit from the entire ventricular system. A block at *A* would produce a unilateral hydrocephalus involving only one ventricle; a block at *S* would produce an internal hydrocephalus involving both lateral ventricles; and to obtain a complete obstruction at the base, the two foramina of Luschka and the foramen of Magendie (*L*, *L*, and *M*) would have to be occluded.

A = foramen of Monroe; *S* = aqueduct of Sylvius; *L*, *L* = foramina of Luschka; *M* = foramen of Magendie.

From a physiologic standpoint, therefore, both varieties of hydrocephalus result from the same cause — a diminished absorption. Although there is an anatomic difference in the patency or closure of the foramina of exit from the ventricles, the absorption is only slightly greater where the foramina are open.

In the previous communication a series of cases of obstructive hydrocephalus was shown in which the pathologic findings verified the clinical evidence of an existing obstruction to the outflow of cere-

TABLE 1.—Cases of Communicating Hydrocephalus Studied with Phenolsulphonephthalein Test

Cases	Age	Absorption After Ventricular Injection		Absorption After Subarachnoid (Spinal) Injection		Communication, Ventricles and Subarachnoid Space; Appearance in Spinal Canal; Ventricular Injection, Min.	History	Postmortem Findings
		Time of Appearance, Minutes	Two-Hour Absorption, per Cent.	Time of Appearance, Minutes	Two-Hour Absorption, per Cent.			
1. R. G.	18 months	30	2.0	..	11.0	1	Definite history of meningitis at 7 months; hydrocephalus followed immediately	Living
		20	2.3	13	7.0	2		
2. M. R.	11 mos. (during meningitis)	13-15	6.5	..	....	7	Meningitis followed by hydrocephalus, which healed spontaneously	Living (spontaneous recovery)
	(during hydrocephalus)	20	0.5	8	14.0-9.5	2		
	9 months later	..	...	8	15.0			
	3 years later	..	...	..	60.0			
3. H. N.	8 months	25	4.4	..	10.0	20	Hydrocephalus noted 6 weeks after birth; acute illness 3 days after birth; meningitis (?)	Living
		25	4.0	..	10.0	13		
4. J. C.	16 months	30	4.0	..	....	1	.....	None
5. W. F.	6 months	..	4.5	..	16.0	3	Congenital.....	Living
6. W. F.	4 months	..	3.0	..	....	2	Convulsion and fever three days after birth	Living
7. E. W.	3 months	..	3.0	..	8.5	1	Meningitis (meningococcus)	None
8. P. G.	8 months	..	5.0	..	10.0	Less than 7 min.	.....	None
9. F. C.	4 months	..	2.0	..	....	7	Meningocele.....	Adhesions at base
10. H. B.	4 months	..	...	..	....	5	Meningocele.....	Adhesions at base
11. F. W.	6 months	16	2.0	12	10.0	Slight in less than 4½ hours.	Meningocele; large head at birth	Adhesions at base
		25	1.0	..	....	None in an observation several weeks later		

brospinal fluid from the ventricles. Since then, numerous other cases have been studied, in all of which an obstruction was invariably found when the phenolsulphonephthalein test indicated such an obstruction during life. It is needless to present these cases in detail, as they merely confirm the previous observations, differing only in the details of the character and location of the obstruction. The accompanying tabulation of these cases (Table 2) gives in condensed form all the fundamental facts concerning the added group of cases of hydrocephalus with obstruction.

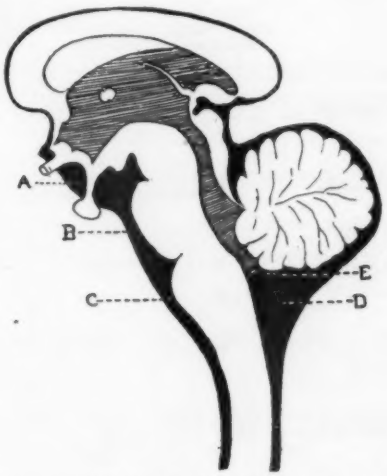

Fig. 2.—Diagram representing the midsagittal view of the brain stem, showing the ventricular system (in cross lines) and the subarachnoid system (in black). The communication between the two systems is at *E* (foramen of Magendie). The foramina of Luschka are not shown.

A = cisterna interpeduncularis; *B* = cisterna pontis; *C*, *D* = cisterna cerebellomedullaris (cisterna magna).

Although a small series of cases of communicating hydrocephalus was studied by the foregoing mentioned methods and presented in the earlier paper, we were unable to obtain any pathologic examinations to explain the clinical findings, and final conclusions on the etiology of this form of hydrocephalus were necessarily deferred, although the conclusions suggested from the tests strikingly anticipated the subsequent pathologic findings. It is largely the purpose of this communication to fill this pathologic deficiency and give the findings at necropsy of four cases of communicating hydrocephalus and to present the pathologic findings which result in the production of this so-called idiopathic disease. All the cases studied are from the services of Professor Halsted and Professor Howland, and it is due to their privileges and continued interest that this contribution is made possible.

I. COMMUNICATING INTERNAL HYDROCEPHALUS

(*a*). *Demonstration of the Communication.*—Eleven cases of communicating hydrocephalus have been studied. In each of these the communication has been demonstrated by the phenolsulphonephthalein test. This is the only absolute method of differentiation of the two

TABLE 2.—CASES OF INTERNAL HYDROCEPHALUS WITH OBSTRUCTION,—

Cases	Age	Absorption After Ventricular Introduction		Absorption After Spinal Introduction		Communication Ventricle and Subarachnoid Space; Time of Appearance
		Time of Appearance, Minutes	Two-Hour Absorption, per Cent.	Time of Appearance, Minutes	Two-Hour Absorption, per Cent.	
1. P. G.	7 months	45	0.75	6	62	None in 45 min.
2. A. H.	6 months	40	1.0	..	..	None in 2 hours
3. N. P.	6 weeks	45	1.0	..	..	None in 20 min.
4. N. M.	2 years	40 35	0.50 0.50	6	35	None in 2 days
5. M. R.	13 months	40 20	0.50 0.9	8	25	None in 1½ hrs.
6. F. W.	6 months	16 25	2.0 1.0	12	10	Trace in less than 4½ hrs. None in 2d observation in 3 hours
7. M. N.	5 months	30	1.5	..	35	None in 30 min. Faint trace in 14 hours
8. R. C.	2 months	..	1.2	..	30	None in 2 hours
9. R. S.	6 weeks	..	2.0	..	..	None in 2 hours
10. L. S.	19 months	..	1.5	..	56	None in 2 hours
11. J. S.	8 months	..	0.5	..	25	None in 2 hours
12. J. B.	3 months	..	0.5	..	..	None in 40 min.
13. A. C.	16 months	..	0.5	..	..	None in 1 hour
14. J. M.	2½ years	..	2.0	..	..	None in 1 hour
15. J. F.	5 years	..	0.5	..	45 41	None in 1 hour

types of hydrocephalus. One can often tell from the amount of fluid obtained by lumbar puncture whether a communication exists, but more frequently this is misleading. If only 3 or 4 c.c. are obtainable on more than one occasion, it is fairly safe to assume that the obstruc-

tion is complete and exists at the foramina of Luschke and Magendie, because all the fluid formed from the choroid plexus is retained in the ventricles. On the other hand, if an obstruction exists at the aqueduct of Sylvius, a large amount of fluid can be obtained by lumbar puncture because the choroid plexus of the fourth ventricle, including

—Studied with the Phenolsulphonephthalein Test

Duration of Excretion		History	Postmortem Findings
Ventricle	Spinal Canal		
3% excreted in 12 hours; three days later, concentration of phenolsulphonephthalein in the urine was diminished	Less than 12 hours	Tuberculous meningitis; meningocele since birth	Exudate over base of brain occluding the foramina of exit
.....	.....	Congenital; myelomeningocele also present	Occlusion of aqueduct of Sylvius
.....	.....	Congenital.....	Total absence of aqueduct of Sylvius
11 days; 6.1 % first 24 hours; 7.5% second 24 hours; 5.7% third 24 hours	21 hours	Congenital.....	Died at age of 5 years. Double obstruction: one at aqueduct, the other at foramina of Magendie and Luschka
.....	.....	Meningitis at 4 months; previously normal	Absence of foramina of Luschka and Magendie. Fourth ventricle a large cyst. Marked thickening of pia-arachnoid
7 days	48 hours	Congenital; syringomyelocele; no history of meningitis	Chronic inflammatory process; adhesions at base occluding foramina of exit
.....	.....	Congenital.....	Living
.....	.....	Congenital.....	Obstruction at foramina of Magendie and Luschka; enormous fourth ventricle, like a huge cyst
.....	.....	Congenital.....	No necropsy
.....	.....	Meningitis lasting one month when 1 year old	Living
.....	.....	Meningitis; congenital....	Very dense adhesions at base of brain, disclosed at operation; child still living
.....	.....	Congenital.....	No necropsy
.....	.....	Acute meningitis.....	Thick exudate of acute meningitis at base of brain
.....	.....	Acute meningitis.....	Living
.....	.....	Tumor.....	Glioma in midbrain occluding aqueduct of Sylvius

the flocculi, is still producing fluid which passes freely into the subarachnoid space. It must be remembered that even in the highest grades of hydrocephalus it is impossible to withdraw more than a certain amount of fluid by needle, either from the ventricle or the sub-

arachnoid space, because a balance between intracranial and extracranial pressure is reached before the ventricles are emptied, and air cannot enter the needle to displace more fluid. False interpretations can be avoided and the existence of communication or obstruction determined with accuracy by the phenolsulphonephthalein test. It should be emphasized again that the ordinary solution of phenolsulphonephthalein is strongly alkaline and produces a marked reaction. A neutral solution which can be used with a minimum degree of danger has been prepared for us by Hynson, Westcott & Dunning. The solution should be freshly prepared.

Phenolsulphonephthalein appears in the spinal fluid in one to three minutes after its introduction into the ventricles where hydrocephalus does not exist. Almost the same obtains in communicating hydrocephalus, although the time in many cases is five to seven minutes, and in one instance was twenty minutes. This increased period was reduced to thirteen minutes by the upright position.

The increased amount of cerebrospinal fluid present in hydrocephalus seems to cause no appreciable delay in the transmission of the dye from the ventricles to the spinal canal. Why the delay should be so much greater in the single instance is not entirely clear, although adhesions along the spinal cord suggest a possible explanation. In cases in which adhesions are present at the base of the brain, the same condition frequently exists along the entire spinal canal, and it is natural to assume that these may delay the descent of the dye.

One patient showed a trace of phenolsulphonephthalein when a lumbar puncture was made four and a half hours after the ventricular introduction, although it was absent one-half hour after its introduction. Several weeks later a similar test was performed and there was a complete obstruction. We must assume a very trivial communication to have existed in this case, and later to have been closed over. At necropsy very dense adhesions totally occluded the foramina of exit from the fourth ventricle. No doubt this cordon of adhesions was becoming progressively tighter and the first phenolsulphonephthalein test caught the condition in the terminal stages of the transition from a communicating hydrocephalus to an obstructive hydrocephalus.

(*b*). *Quantitative Absorption in Communicating Internal Hydrocephalus.*—There is very little to add to our previous report on the quantitative absorption of cerebrospinal fluid except the confirmation of additional material. The variation of the amount of absorption is within narrow limits; the average is 3 to 4 per cent. during a two-hour period, the highest 5 per cent., and the lowest 0.5 per cent. The absorption following introduction into the normal ventricle is 12 to 20 per cent. When introduced into the spinal canal the variation is

also small. The average two-hour absorption is 10 per cent., the highest 16 per cent., the lowest 7 per cent., the normal 35 to 60 per cent. The time of first appearance in the urine is also greatly delayed following either intraventricular or intraspinal injection.

One interesting case has been under observation for four years. The child was first seen with an acute epidemic cerebrospinal meningitis, and the absorption tests showed a moderate delay and diminution in absorption following the ventricular test. The ventricles were shown to communicate with the subarachnoid space. A few weeks later, after the subsidence of the acute meningitis, the child was again brought to the hospital apparently becoming blind and having an

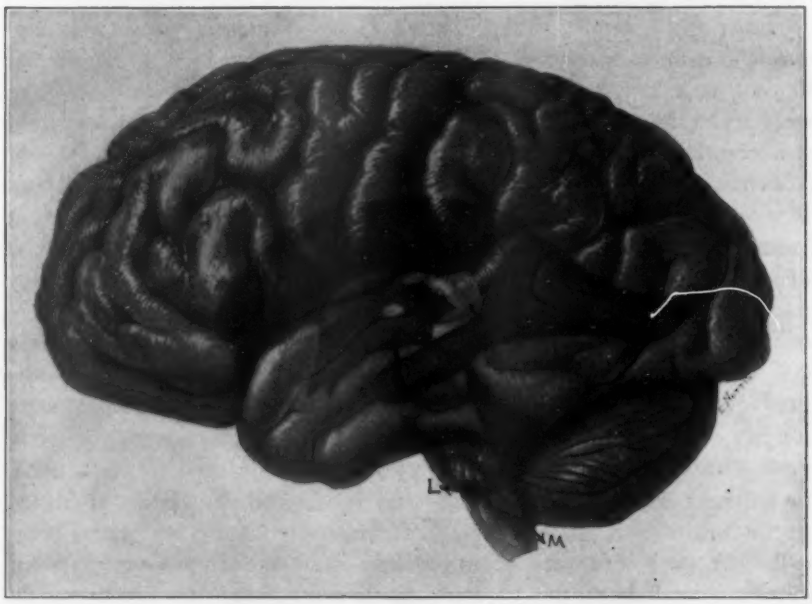

Fig. 3.—Plastic representation of a lateral view of the ventricular system, to show the relation of the structures of the brain (modified from Gray's Anatomy). *L* = foramen of Luschka; *M* = foramen of Magendie.

enlarged head, with a bulging tense anterior fontanel. Large amounts of cerebrospinal fluid were obtainable both from the ventricles and the subarachnoid space. The condition was obviously internal hydrocephalus, which was shown to be of the communicating variety. The absorption from the ventricles (0.5 per cent.) and subarachnoid space (14 and 9.5 per cent. on two occasions) was characteristically low. Phenolsulphonephthalein appeared in the spinal canal two minutes after its introduction into the ventricles. The condition seemed hopeless, and the parents were so instructed. Much to our surprise, four

years later the patient again appeared, with a fractured skull, but the hydrocephalus had apparently cleared, and the child on discharge appeared entirely normal. Tests at this time showed 50 per cent., or a normal phenolsulphonephthalein output from the subarachnoid space. The roentgenogram of the skull was negative.

(*c*). *Pathological Findings in Communicating Internal Hydrocephalus.*—A pathologic examination has been obtained in four cases of communicating hydrocephalus. Three of these have been studied in the above series and the fourth is a specimen which has been in the pathologic museum for several years. Because of the similarity of the findings in all cases, the results will be grouped and individual mention made only of accessory details.

Each of the four cases presented exactly the same pathologic condition — a barrier of very dense adhesions at the base of the brain. In each case the foramen of Magendie and one foramen of Luschka were sealed by adhesions and the other foramen of Luschka was patent to a certain degree, and through this single channel the fluid escaped from the fourth ventricle and therefore from the ventricular system. The adhesions completely encircled the brain anterior to the patent foramen of Luschka; and the basal cisternae — cisterna magna, cisterna pontomedularis, and cisterna interpeduncularis—were completely obliterated by these adhesions. The adhesions in each instance were most dense in the region of the medulla and cerebellum and became less pronounced in the chiasmal region. Minor adhesions were scattered over the surface of the brain. In one case the adhesions were present over the base, with additional dense adhesions completely binding the tentorium to the posterior surface of each occipital lobe, the superior surface of both cerebellar lobes, and completely encircled the midbrain as it passed through the opening in the tentorium cerebelli. In each instance an apparently complete encircling mass of adhesions sealed off the base of the brain so that the cerebrospinal fluid could not pass forward to the cerebral subarachnoid space, but only downward into the spinal canal. The obliteration of the various basilar cisternae, which are the centers from which cerebrospinal fluid is distributed over the cerebral subarachnoid space, may in itself be sufficient to eliminate the cerebral subarachnoid space from absorption, even without the encircling adhesions.

No doubt in many cases both foramina of Luschka may be obliterated, with a patent foramen of Magendie, or vice versa. In any case, the communication between the ventricles and subarachnoid space, though limited to one foramen at the base, is ample, being at least larger than the aqueduct of Sylvius. After passing from the ventricles, however, the absorption is limited to the *spinal* subarachnoid space and a minimal area of the subarachnoid space around the cerebellum.

The phenolsulphonephthalein tests show a reduction in absorption from the subarachnoid space to about one-fourth or one-fifth of the normal. This roughly corresponds to the diminution of the subarachnoid absorbing area due to the elimination of the cerebral subarachnoid space by adhesions.

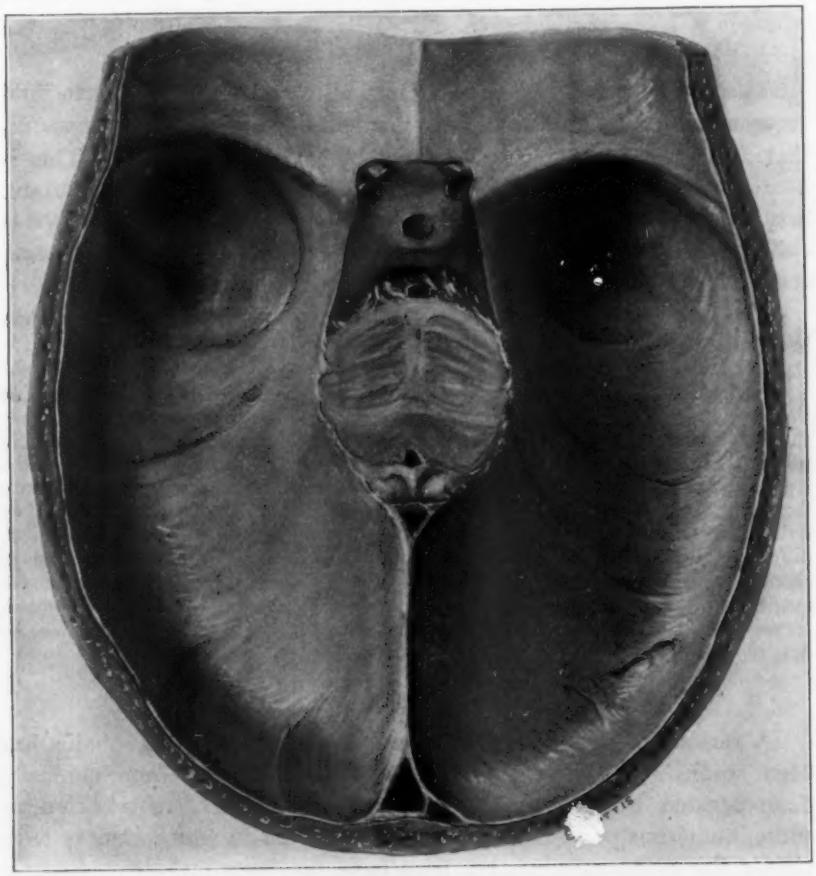

Fig. 4.—View of dura, showing midbrain passing from the posterior to the anterior fossa through the opening in the tentorium.

Note the adhesions between the periphery of the midbrain and the tentorium, also the dura along the base of the skull. The communication between the posterior and middle intracranial fossae is destroyed by these adhesions, and when this is true a communicating internal hydrocephalus results.

In each instance the adhesions between the brain and the dura were dense, necessitating their incision to prevent tearing the cortex during the removal of the brain. Special emphasis should be placed on the necessity of demonstrating the adhesions during the progress of the postmortem examination, for after they have been separated their

demonstration becomes difficult. This is especially true when the adhesions are thin and toward the periphery of their distribution. One not infrequently sees a large area of adhesions over the cerebral cortex which causes no internal hydrocephalus. This is probably due to the fact that the base is the distributing center for all the fluid that covers the brain, and the closure of the cisternae prevents the distribution, whereas a large cortical area can easily be compensated for by the normal excess of the absorbing area.

The presence of adhesions is responsible for the fact that fluid accumulates in the brain and not outside the brain, or, in other words, that an internal and not an external hydrocephalus results. This is because the brain is tightly bound to the dura, and more important, because the fluid-containing and fluid-absorbing subarachnoid space is obliterated over most of the brain, and especially at the distributing center in the cisternae.

The truth of the following paragraph from our former paper has been susbtantiated by the results of the pathologic examinations:

> We have had no pathologic examination on patients with hydrocephalus of the communicating type. It is very likely that the diminished absorption from the subarachnoid space is due to adhesions which diminish the size of the subarachnoid space. Adhesions anterior to the foramina of Luschka, by causing obliteration of the cisterna magna, would prevent the passage of fluid into the general cerebral subarachnoid space as effectually as if the aqueduct of Sylvius were obliterated. The two groups would then be essentially similar, differing only in the fact that the spinal subarachnoid space participated in absorption in the communicating type. . . . How much alteration in the meninges alone, without adhesions, interferes with absorption, cannot be stated. It seems to us probable that the major part if not all of the disturbance is due to the limitation of the subarachnoid space.

II. OBSTRUCTIVE INTERNAL HYDROCEPHALUS

A series of fifteen cases of obstructive internal hydrocephalus has been studied, in each of which the obstruction has been clinically demonstrated by the phenolsulphonephthalein test. In addition to these, numerous other cases have been observed in which tumors have caused the hydrocephalus, but they have not been included. Several other cases have been seen at necropsy, but are also not included because they were not previously observed clinically. In ten of these fifteen cases the obstruction has been verified by a postmortem examination. One was due to an acute tuberculous meningitis which sealed the foramina of Luschka and Magendie. Another case was an acute cerebrospinal meningitis in which the base was covered by exudate. Four cases had complete occlusion of the aqueduct of Sylvius, three of which showed histologic epithelial remnants of this structure, and the fourth was due to a solitary tubercle in the midbrain which had grown into and totally occluded the aqueduct of Sylvius. One of these

cases with iter obstruction had a second obstruction by adhesions, completely closing both foramina of Luschka and the foramen of Magendie. This child (N. M. 4) lived five years and was under observation most of that time, and despite an enormous hydrocephalus, did not lose all evidences of intelligence. At necropsy there was in

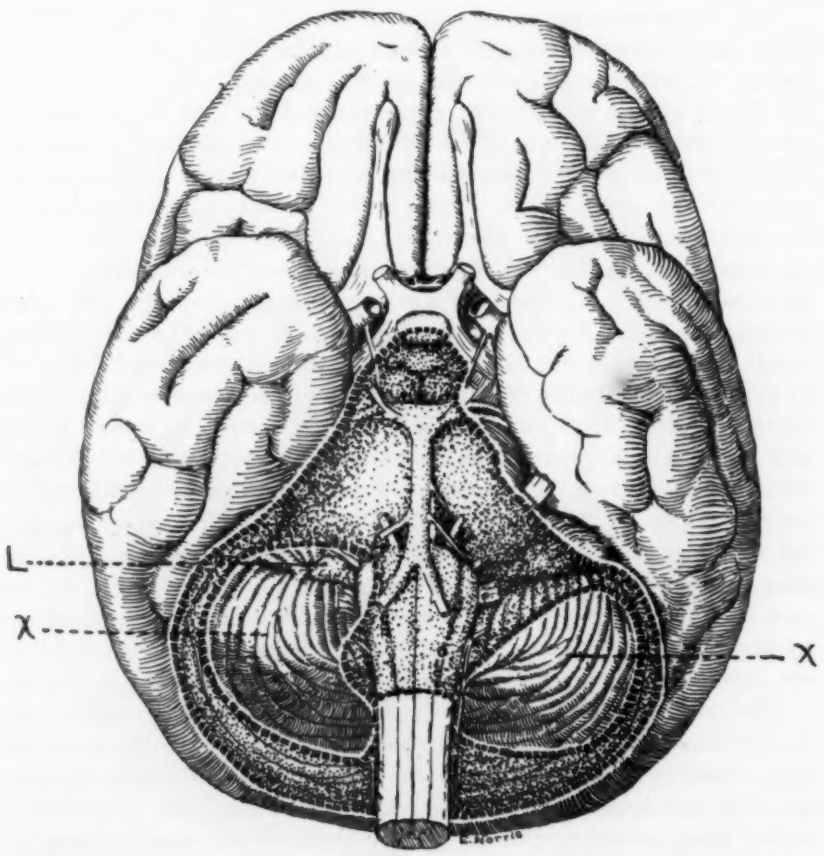

Fig. 5.—Diagram of the base of the brain, showing in diagram form, roughly, the distribution of the adhesions over the base of the brain responsible for the communicating type of internal hydrocephalus.

X, X represent areas on the cerebellar hemispheres which are relatively free from adhesions.

L represents the foramen of Luschka, which is patent only on this side. The opposite side is completely obliterated by adhesions. The adhesions extend along the tentorium on both the superior and inferior surfaces.

addition to the hydrocephalus a congenital urethral obstruction and a double hydronephrosis. The brain presented a great dilatation of the third and lateral ventricles anterior to an iter obstruction, and a dilatation of the fourth ventricle, anterior to the closure of the basal

foramina of exit. Adhesions between the cerebellum and dura no doubt restricted the dilatation of the fourth ventricle and mechanically prevented its enlargement into a great cyst. It was impossible to have suspected the presence of the second or iter obstruction. The obstruction at the base was certain because of the very scant amount of cerebrospinal fluid (2 to 4 c.c.) obtainable at successive lumbar punctures.

(*a*). *Demonstration of the Obstruction.*—In each of these fifteen cases, phenolsulphonephthalein failed to appear in the spinal fluid following its introduction into the lateral ventricle. In one instance (F. W. 6) a trace was present, appearing at some time in the interval between one-half and four and a half hours. Several months later the obstruction had become complete and phenolsulphonephthalein did not appear in the spinal canal. There was a corresponding reduction in the absorption from 2 to 1 per cent., and a corresponding increase in the appearance time. The significance of these observations will be mentioned later, in considering the interrelationship of the two types of hydrocephalus. In each of the fifteen cases the output of cerebrospinal fluid was under 2 per cent. as compared to the normal of 15 to 20 per cent., and readily explains the cause of the hydrocephalus. The spinal absorption in several cases was entirely normal; in several it was quite low, 10 to 30 per cent., as compared to the normal of 35 to 60 per cent. In these cases the amount of absorption is really immaterial in the production of the hydrocephalus, because the fluid never reaches the meninges. Its importance, however, is in the prognosis following operative intervention, for with a low spinal absorption (10 to 30 per cent.) the release of the obstruction would then transfer an obstructive hydrocephalus to a communicating hydrocephalus, and only modify but not cure the disease. It would only make its development less rapid.

The total time for elimination of the dye after ventricular introduction was studied in only a few cases, in each of which the excretion was carried over a period of several days; in one case eleven days on two different occasions. The excretion following spinal introduction varied from normal to two days, an index of which time is given by the two-hour output in the urine.

COMPARISON OF THE TWO TYPES OF HYDROCEPHALUS

Both types of hydrocephalus are due to a diminution in the absorption of cerebrospinal fluid. Both are obstructive, differing only in the location of the obstruction. In the so-called obstructive variety, the obstruction is within the ventricular system. In the so-called communicating hydrocephalus, the obstruction is in the subarachnoid space. In communicating hydrocephalus the obstruction is probably always the result of adhesions, which, of course, means that an inflammatory process has existed before birth if the hydrocephalus is congenital, or

it may develop at any time after birth. Such an inflammation of the meninges may seal the three foramina of exit from the fourth ventricle and produce an obstructive hydrocephalus, or it may leave one, two, or all the foramina patent and seal off the cisterna, producing the communicating type of hydrocephalus; or it is possible to do both; that is, the foramina may all be closed and the meninges sealed anterior

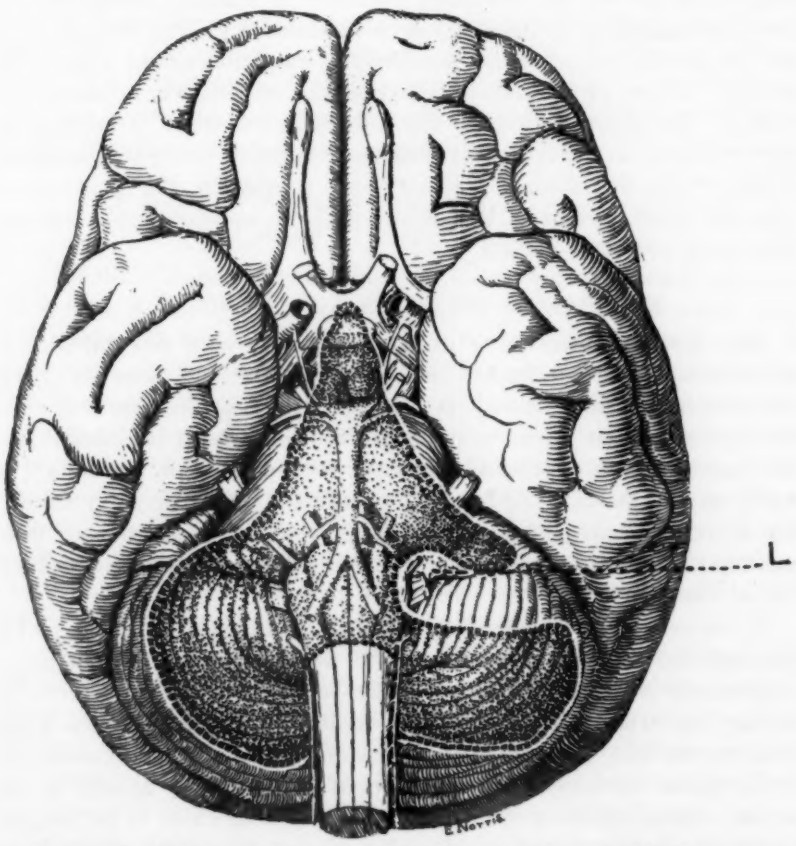

Fig. 6.—A diagram of the base of the brain showing roughly the extent of the adhesions at the base.

L is the patent foramen of Luschka, on one side only. The other foramen of Luschka and the foramen of Magendie are completely obliterated by adhesions. In this case the adhesions did not involve the tentorium, but only the base of the brain.

to the foramina. This, of course, would produce the obstructive hydrocephalus, and the meningeal obliteration, though of no consequence (because the fluid does not reach the meninges) could be demonstrated clinically only by tests as a greatly reduced absorption from the subarachnoid space. Case F. W. 6 is such an instance of a

combination of the two types. At first there was a minute and greatly delayed communication. The process was undoubtedly progressive, as later the obstruction became total and the absorption following ventricular introduction correspondingly diminished. In all of our cases of communicating hydrocephalus the foramen of Magendie has been closed, together with one foramen of Luschka. A gradual reduction in the size of the remaining foramen of Luschka would change the communicating hydrocephalus to obstructive hydrocephalus, as in the foregoing case, whereas clinically the transition would scarcely be noticed. It is, of course, of the greatest importance to determine whether the absorption from the subarachnoid space is normal or approximately so before attempting any operative procedure designed to relieve the obstruction. As mentioned previously, the transference from an obstructive to a communicating hydrocephalus would add but little to the patient's welfare.

MENINGOCELE AND INTERNAL HYDROCEPHALUS

The frequent association of a meningocele with internal hydrocephalus has long been known, and quite frequently an internal hydrocephalus has been observed to develop following the removal of a meningocele. This seems to us most probably due to the removal of the absorbing area of the meningocele, which was just sufficient (or nearly so) to maintain the balance between the production and absorption of cerebrospinal fluid. With its removal the production of fluid becomes sufficiently greater than absorption to cause increased dilatation of the ventricles.

In eleven patients with communicating hydrocephalus, three had a meningocele; and in fifteen cases of obstructive hydrocephalus, a meningocele was present in three. The formation of a meningocele in conjunction with a communicating hydrocephalus, arising in early intra-uterine life, seems explainable by the pressure which causes the cerebrospinal fluid to take the path of least resistance, usually in the lumbar region, which is the last part of the spinal canal to be bridged over. The presumption is natural that a meningocele signifies an increased pressure of cerebrospinal fluid in the spinal canal, or a communicating hydrocephalus. But that this is not necessarily true is shown by its existence in three cases of obstructive hydrocephalus. In one, however, the hydrocephalus was originally of the communicating variety, and when first seen by us was in the last stages of the transition to a total obstruction. The other case was a myelocystocele, the cerebrospinal fluid having no communication with the sac, which was now blind, and even the original communication with the central canal of the spinal cord was closed in several places. The relationship of the meningocele to the obstructive hydrocephalus in the remaining

case could not be ascertained because of the absence of a pathologic examination.

In all cases with a meningocele which have recently come under our observation, careful studies have been made of the amount of absorption from the spinal canal in addition to other evidence of internal hydrocephalus. Only in those cases in which a normal absorption is present from the spinal canal have we removed the meningocele, and in these cases have observed no subsequent secondary effects on the brain. The phenolsulphonephthalein output from the spinal canal we regard as a thoroughly reliable index of the function of the meninges, and know of no other way in which a safe decision can be made in the operative treatment of a meningocele.

THE RELATION OF MENINGITIS AND OTHER FACTORS TO INTERNAL HYDROCEPHALUS

It is our opinion, based on the clinical studies and pathologic findings, that the great majority of cases of hydrocephalus result from meningitis. A hydrocephalus of short duration may occur during the meningitis, due to the tubercle bacillus, the pneumococcus, and doubtless other organisms. Chronic hydrocephalus is to be expected only following a meningococcus meningitis. That a very mild form of what was probably meningococcus meningitis has been the cause of a number of our cases seems evident from some of the history, but the illness has been so slight and recovery so prompt that the mother has looked on it as a "cold," "stomach trouble," or some illness incident to teething, and it is only by careful questioning that the illness is recalled. Yet after these mild symptoms hydrocephalus may result.

At necropsy adhesions are found which have the usual basal distribution of other types of meningitis, and these adhesions are the cause of the internal hydrocephalus. At necropsy exactly the same findings are obtained in cases which have an internal hydrocephalus at birth; that is, adhesions over the same general distribution, affording proof of the existence of an intra-uterine inflammatory process. What organism is responsible for these lesions is entirely unknown. So is also the organism that causes intra-uterine lesions of other serous membranes. In no instance have we been able to elicit any illness of the mother occurring during gestation which could account for the meningitis in the child.

An analysis of the accompanying table (Table 3) shows the high percentage of cases in which meningitis is the basis for the hydrocephalus. The figures are intended only to give the results from a small group of cases and may no doubt be greatly altered with increasing numbers. Both types of hydrocephalus are caused by this process, as heretofore mentioned. It is really difficult to understand how the

TABLE 3.—SUMMARY OF ETIOLOGIC FACTORS IN CASES OF INTERNAL HYDROCEPHALUS

Hydrocephalus	Meningitis			Congenital Malformation; Not Inflammatory	Tumor	Uncertain Whether Congenital or Inflammatory Process; Foramina at Base Occluded	No Proof of Character of Lesion Because no Examination
	Clinical Alone	Necropsy or Operation Alone	By History and Operation or Necropsy				
Obstructive	2	2	3	3	1	1	3
		46.6%		(20%)	(6.6%)	(6.6%)	(20%)
Communicating	4	3	..	..	..	..	4
		63.6%					(37.4%)

Excluding those cases in which no evidence is obtainable because of absence of examination by operation or necropsy:

1. The percentage of meningitis cases in obstructive hydrocephalus is 58.3 per cent.
2. The percentage of meningitis cases in communicating hydrocephalus is 100 per cent.
3. The percentage of meningitis cases in both obstructive and communicating hydrocephalus is 80 per cent.
4. The percentage of congenital malformation causing hydrocephalus (in all cases) is 15.8 per cent.; and excluding communicating hydrocephalus, is 25 per cent.

communicating hydrocephalus can be caused by any other process than a meningitis, because of the rather diffuse area necessary to be invaded to produce these results. In the series of eleven cases of communicating hydrocephalus, four gave a definite history of meningitis, but from these no anatomic examinations were possible; three others gave no history of meningitis, but the condition was evidently congenital, because each had a lumbar meningocele and at necropsy each of these gave all the evidences of an old extensive basilar meningitis. Of the four remaining cases in this group, three were evidently of congenital origin, the enlargement of the head being noticed very shortly after birth, and the fourth was noticed when the child was 3 months old and may also have been congenital, no illness having been noticed between the time of birth and the enlargement of the head. As an anatomic examination was not made in any of these four cases, the etiology remains entirely obscure.

Two cases of obstructive hydrocephalus (M. R. 4 and R. C. 8) present an almost indistinguishable anatomic picture. In each patient the foramen of Magendie and both foramina of Luschka were entirely absent, and the posterior fossa was filled with a cyst, resulting from a dilated fourth ventricle. The other ventricles were proportionately dilated. In one of these cases the child was perfectly normal until four

months old, when a typical mild meningitis developed. The hydrocephalus followed immediately. At necropsy only a few minor adhesions were found. Had not the history been definite, the condition could easily have been regarded as congenital and the foramina at the base as congenitally absent, but the condition was unquestionably due to the meningitis, few traces of which remained. The other case with a similar anatomic picture was of congenital origin and the foramina were likewise closed. Whether an intra-uterine inflammatory process caused the condition similar to the extra-uterine meningitis of the other case, or whether the foramina were congenitally absent, could not be decided.

Congenital obliteration of the aqueduct has been adequately presented in the previous paper. Recently, Schlapp and Gere[1] presented additional cases of the same character.

One tumor has been included in this series. Tumors, of course, with rare exceptions, produce only the obstructive type of hydrocephalus. In infancy they are extremely rare and it is only in early childhood that this becomes an important factor to consider. As age increases hydrocephalus becomes more the result of tumor and relatively less the result of meningitis, on account of the increasing frequency of tumors.

SPONTANEOUS CURE OF INTERNAL HYDROCEPHALUS

Spontaneous recovery is not infrequent in internal hydrocephalus. This is evident from the cases seen from time to time of arrested growth of the abnormally large head. We have at least three cases under observation in which the diagnosis is clear, and in which there has been no change in the size of the head of the patient for years. Another child was studied through an attack of meningitis into the stage of hydrocephalus. After being lost sight of for three years, she returned apparently entirely cured. Many patients with meningitis recover without an internal hydrocephalus, and no doubt many cases of hydrocephalus follow meningitis and sooner or later recover.

Spontaneous recovery naturally depends on the cause of the disease. A spontaneous cure in a case of hydrocephalus with a congenital or other obstruction at the aqueduct seems entirely impossible, but it is not difficult to imagine recovery following the gradual disappearance of adhesions. This is especially true when one realizes how adhesions may disappear. The reverse may also be true and adhesions in the meninges as elsewhere may be progressive and increase the hydrocephalus. It is also possible to imagine a sudden rupture of the thin

1. Schlapp, M. G., and Gere, B.: Occlusion of the Aqueduct of Sylvius in Relation to Internal Hydrocephalus. Am. Jour. Dis. Child., 1917, **13**, 461.

wall of a large fourth ventricle cyst, producing a new foramen of exit to the subarachnoid space. It is not improbable that the majority of cases having a meningocele but no hydrocephalus, represent a regressive stage or cure of an old intra-uterine hydrocephalus. Otherwise it is difficult to understand the formation of many meningoceles.

SUMMARY AND CONCLUSIONS

1. Twenty-six cases of internal hydrocephalus have been studied, fifteen of the obstructive and eleven of the communicating variety.

2. These cases have been studied with intraventricular and intraspinal injections of phenolsulphonephthalein.

3. Postmortem examinations have demonstrated an obstruction in every case in which an obstruction has been shown clinically by this test. The obstruction may be a congenital malformation or inflammatory process or tumor, and occur at any part of the ventricular system, but usually at the aqueduct of Sylvius or the foramina of Luschka and Magendie.

4. In all cases of obstructive hydrocephalus there is practically no absorption from the ventricles, and frequently (although not necessarily) a normal absorption from the subarachnoid space. Hydrocephalus results because the fluid is mechanically prevented from passing from the ventricles, where the fluid forms, to the subarachnoid space, where it is normally absorbed, and where only it can be absorbed.

5. Communicating hydrocephalus is caused by a barrier of adhesions at the base of the brain which mechanically prevents the cerebrospinal fluid from reaching the cerebral subarachnoid space, where the greatest part of absorption normally takes place. The various cisternae or centers for the distribution of cerebrospinal fluid are more or less obliterated by adhesions.

6. Absorption of cerebrospinal fluid is a general process, from the entire subarachnoid space, and communicating hydrocephalus results because only a fraction of this area can be utilized for absorption.

7. These pathologic findings harmonize with the clinical phenolsulphonephthalein tests, which show a greatly diminished absorption from the subarachnoid space.

8. Obstructive and communicating hydrocephalus are, therefore, essentially the same, and in reality all are due to obstruction. In the obstructive variety the obstruction is in the ventricular system; in the communicating variety the obstruction is in the subarachnoid space.

9. Obstructive hydrocephalus may, by operation or spontaneously, change to communicating hydrocephalus, or the reverse may occur spontaneously. Careful studies with the phenolsulphonephthalein test will indicate these possibilities.

10. Meningitis is by far the greatest etiologic factor in both types of hydrocephalus and probably always causes the communicating variety. This may be either prenatal or postnatal. The meningitis may be of a very mild grade and easily overlooked.

11. There is a definite relationship between a meningocele and internal hydrocephalus; both usually probably result from the same cause. The removal of a meningocele may aggravate a fairly well balanced or even arrested case of internal hydrocephalus. This probably results from a diminution of the absorbing area.

12. Spontaneous recovery of internal hydrocephalus sometimes occurs.

13. We feel that the surgical treatment of internal hydrocephalus has now a definite anatomic basis and hopeful prospects. The operative results of a series of cases will appear in a subsequent communication.

A MENTAL AND PHYSICAL SURVEY OF A GROUP OF JUVENILE DELINQUENTS

WITH NOTES ON THE YERKES-BRIDGES POINT SCALE *

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At the request of the Boys' and Girls' Aid Society of San Francisco we undertook in September, 1916, a study of the boys placed in the society's care by the various juvenile courts of California. A few of these were committed as orphans or destitute, but the great majority were committed to detention because of truancy, incorrigibility or active crime.

Our study had for its primary purpose the disclosure of physical and mental defects, but was later extended, with the assistance of Miss Annette Rosenshine, to an investigation of the home conditions of the mentally normal or dull normal boys.

We have also taken this opportunity to compare the Yerkes-Bridges Point Scale with the Binet-Simon Scale (1911 Goddard revision) and to compare the physical measurements with the mental tests, following in some respects the recent work of Doll.

Our physical examinations were made according to the routine of the Stanford Children's Clinic, and included investigations of the skin, eyes, ears, teeth, tonsils, pharynx, thyroid gland, lungs, heart, abdomen, genitals and extremities; the tendon reflexes, and the posture. In addition, vision was tested by the Snellen card at 20 feet without glasses, and a rough estimate of hearing was obtained by the watch test. The Wassermann test was applied in sixty cases, including about half of those in which enlarged epitrochlear glands were found, but owing to circumstances not under our control, could not be carried out in the remainder. Measurements of standing height, chest circumference, head circumference, and of the grip of each hand (dynamometer) were made. It is a matter of regret that the inconvenience of transporting our spirometer to and from the home of the society made it impossible to obtain measurements of vital capacity.

GENERAL PHYSICAL ABNORMALITIES OF THE GROUP AS A WHOLE

Taken as a whole, the 110 boys first examined were somewhat below the normal in height, with a percentile[1] of 44, as compared with the normal average of 50. The percentile for weight, however, was

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* From the Department of Pediatrics, Stanford University Medical School.

1. All percentiles were calculated from the Smedley tables, in which the group average is 50, with a minimum normal of 0 and a maximum normal of 100.

57 and the percentiles for right and left grip were 55 and 59, respectively, all of which are well above the normal. The superiority of the left grip is noticeable. Of the boys, 18.4 per cent. showed vision below 20/30 for the right and 25.2 per cent. for the left eye. Strabismus — convergent except in one instance — was found in 10 per cent. Defective hearing occurred in 15.4 per cent. The teeth were decayed in 53 per cent., irregular or maloccluded in 7.3 per cent. and merely dirty in 11.2 per cent. The tonsils were enlarged in 36.4 per cent. and had been removed in 11.7 per cent. Enlargement of the thyroid gland was not encountered. The anterior cervical glands were palpable in 61 per cent., the posterior cervical in 50 per cent., the inguinal in 74.5 per cent., the axillary in 50 per cent. and the epitrochlear in 19.1 per cent. Wassermann tests were made in ten of the twenty-one cases in which the epitrochlears were palpable without a single positive reaction being obtained. In the one case, out of the sixty tested, in which a positive reaction did occur, the epitrochlears were not palpable. Our respect for this time-honored sign of syphilis has been lessened. Abnormalities of the chest, noted as "flat," "narrow," "pigeon-breasted," occurred in 10 per cent. Two cases of chronic endocarditis were found. One inguinal hernia was discovered. The prepuce was elongated or adherent in 21.7 per cent., and had been circumcized in 20.9 per cent. Four instances of unilateral cryptorchidism were found. Pubic hair was present in 54.5 per cent. Disease of the skin — acne, furunculosis or impetigo — occurred in 10.9 per cent. The knee jerks were unusually lively, without other signs of spasticity, in 6.4 per cent. Postural defects — round shoulders, wing scapulae, lateral curvature of the spine — were noted in 22.7 per cent.

Taken as a whole, and leaving aside for future consideration the various measurements and certain special features, the physical defects are those common to any group of children whose hygiene has been long neglected.

MENTAL EXAMINATION

At the time when the present study was begun, the children's clinic was considering the merits of the Yerkes-Bridges[2] Point Scale, and for purposes of comparison a majority of the boys, ninety-eight in all, were examined both by the Binet-Simon Scale (1911 Goodard revision) which had been used hitherto in the clinic, and by the Yerkes-Bridges Scale.

The Yerkes-Bridges Point Scale was devised by Robert M. Yerkes, assisted by James W. Bridges, following a suggestion by the late Dr. E. B. Huey. A complete description of it was published in 1915 by Yerkes, Bridges and Hardwick.[2] The purpose of the authors was to construct a new scale to

2. Yerkes, R. M., Bridges, J. W., and Hardwick, R.: A Point Scale for Measuring Mental Ability. Baltimore, Warwick and York, 1915.

supplant the Binet-Simon Scale and its various revisions, which had been found by many workers to be unsatisfactory. They included, however, a considerable number of the Binet tests in their own method. The difference between the two scales will be best indicated by a brief description of each.

The Binet Scale, as well as its revisions, presents a series of tests arranged in order of presumably increasing difficulty, and divided in groups of four or five, each group corresponding with a year of "mental age." The mental age of the subject of the examination is that corresponding to the highest complete group of tests that he passes, plus the figure for the number of tests, expressed as fifths of a year, that he passes in the higher groups. Thus, if a child passes all the tests up to and including Group X, three tests in Group XI, and one test in Group XII, his mental age is stated as 10⅗ (10 + ⅗ + ⅕). In this scheme it is theoretically assumed that (1) each group of tests examines satisfactorily for the age the various mental functions; (2) that every "normal" child without regard to environment or training can answer all the questions assigned to his physical age; (3) that inability to answer perfectly any question or test implies complete failure in that test; (4) that all tests are of mathematically equal weight in evaluating intellectual capacity. As a matter of fact—and this is an important objection to the Binet system—every examiner is compelled to modify to some extent the result of every examination by taking into account certain extraneous factors, such as language difficulty, unfavorable environment, and so on. The personal equation, therefore, enters into all Binet testing, and to an undesirable extent.

The Point Scale presents a series of tests, arranged roughly (for the sake of convenience only) in order of difficulty but not divided into age groups, which are designed to test the various mental functions, such as auditory memory, motor coordination, ideation, kinesthetic discrimination, logical judgment. To each of these tests a certain value, expressed in points, is attached. The sum of these values, that is, the score attainable by completion of all tests, is 100. The score attained by the individual examined is the sum of the points assigned to the tests in which he is successful. Part credit is given for partial completion of the tests, thus abandoning the all-or-none principle of the Binet system. The final score is then compared with the norm. This norm is properly made a variable quantity, allowance being given for favoring or unfavorable circumstances of environment and training, factors which practically all investigators recognize as influencing the capacity of individuals to pass mental examinations. The "setting" of the norm, however, can be determined by a direct comparison of the scores of other individuals belonging to the same group and need not be left to the personal judgment of the examiner. The Point Scale offers both an absolute scale of measurement—in relation to an ideal 100—and a relative scale—in relation to the norm for the particular social or educational group to which the individual belongs. The improvement over the older scale is thus in the direction both of objectivity and of flexibility. As the authors point out, the results of examinations at the hands of different examiners are directly comparable, and at the same time the norms can be constantly corrected by experience and comparison.

The discrepancies between the two methods of examination, shown graphically in Chart 1, are considerable and of rather serious significance. In adopting norms for the Point Scale it must be emphasized that we do this only for comparison with the Binet Scale, and that, to our mind, norm-comparison is not the chief purpose of the Point Scale, which has its greatest value rather in estimating total mental capacity. The point scale, as has been pointed out by its originators, has the advantage of being self-perfecting and is not committed to

rigid norms, so that we have taken the liberty of modifying the original figures of Yerkes and Bridges by comparison with those of Haines,[3] derived from a study of individuals belonging to about the same social stratum as the one with which we were dealing, and with data of our own. The norms for ages above 15 cannot be regarded as fully established: those we have adopted are based partly on the Yerkes-Bridges figures and partly on data of our own, and must be taken as only approximations, with an error of perhaps 3 per cent.

We suggest that the originators of the point scale are rather too conservative in stating that the scale is not of great value above age 16. We have a conception, which may or may not be correct, that the scale tests with considerable completeness and accuracy what we may call basic intelligence. By this we mean the broad foundation of

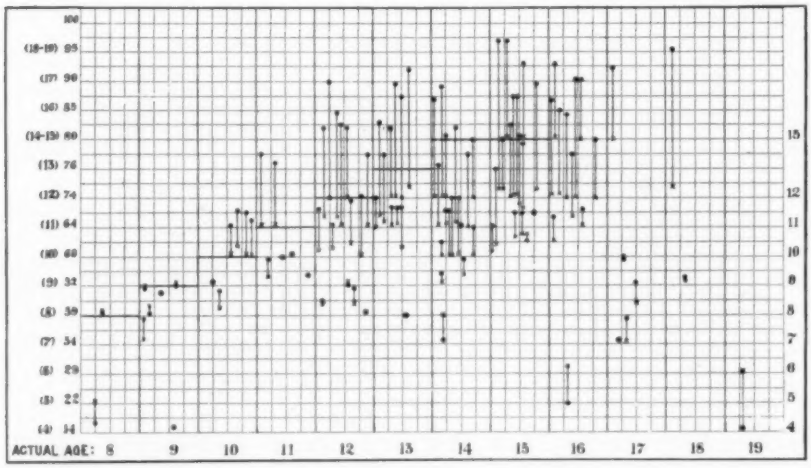

Chart 1.—Comparison of the Yerkes-Bridges Point Scale score and the Binet age of ninety-eight delinquent boys. The Point Scale score and Binet age of each boy are connected by a vertical line. Point Scale score = ●; Binet age = ×.

mental endowment which every normal person must possess before he can branch out into the more highly specialized, so-called higher intellectual functions of adult life. Above this basic level, which is common to all normal individuals, mental growth is along the line of special acquirement and follows closely educational direction. Therefore we feel that so-called adult tests are rather impracticable except along special lines, but that basic intelligence can be tested at any age and is of value at any age.

Our Binet tests — Goddard's 1911 revision with tests for age 15 and for the adult — were practically useless above age 12, and were

3. Haines, T. H.: Mental Examination of Delinquent Boys and Girls. Illinois Med. Jour., 1915, **28**, 283.

also very defective for age 11. This is in accordance with the experience of most investigators. It is plain from Chart 1 that a Binet age of 12, or 12 and a fraction, may represent widely varying degrees of intelligence, as measured by the Point Scale. That the Goddard tests for age 15 and for the adult fail to remedy the fault is also clear.

In Chart 2 we have compared all our scores (calculated as intelligence quotients[4]) by the two scales, arranging them in an ascending order of the Point Scale intelligence quotient. Up to 0.90 the discrepancies nearly balance, the Binet quotient being higher in 17, and the Point Scale higher in 19. At 0.65 and below (P. S. I. Q.) (that is, in the unquestionably defective group) nine Point Scale quotients are lower, and only two Binet quotients are lower, two being the same. Above 0.90 (P. S.) *only one* Binet quotient was higher (by 0.03).

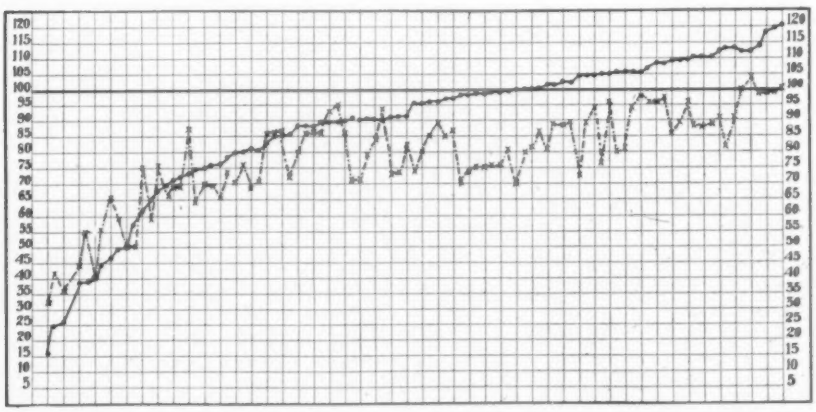

Chart 2.—Comparison of all Point Scale and Binet intelligence quotients arranged in ascending order of Point Scale quotients. In this and following charts ● = Point Scale intelligence quotients; × = Binet Scale intelligence quotient.

Considering a Point Scale quotient of 1.10 or more as evidence of mental superiority, we find in this group that the Binet quotient gave no indication whatever of such superiority; three out of five cases with the highest P. S. quotients were actually below 1.00 by the Binet, and only one was above 1.00.

Charts 3 and 4 are arranged to show the degree of "overlapping" between groups by the two scales. Kohs[5] has already drawn attention to this phenomenon. We have provisionally adopted a maximum of 0.95 for the dull normals, instead of 0.90 as generally used for the

4. The intelligence quotient equals the mental age divided by the actual, or chronological, age.

5. Kohs, S. C.: The Borderlines of Mental Deficiency. Jour. Psycho-Asthenics, 1916, **20,** 63.

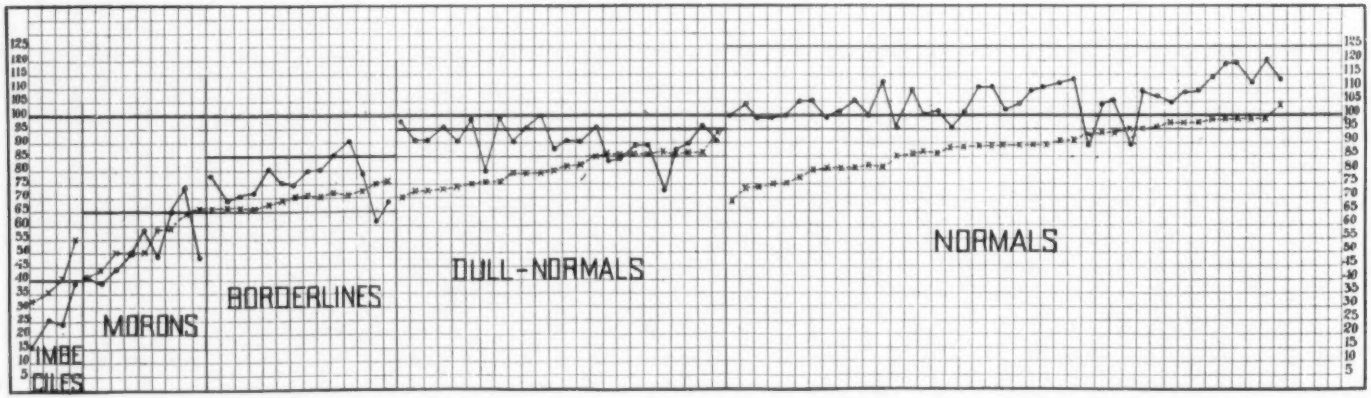

Chart 3.—Comparison of Binet Scale and Point Scale quotients arranged on the basis of Binet quotient.

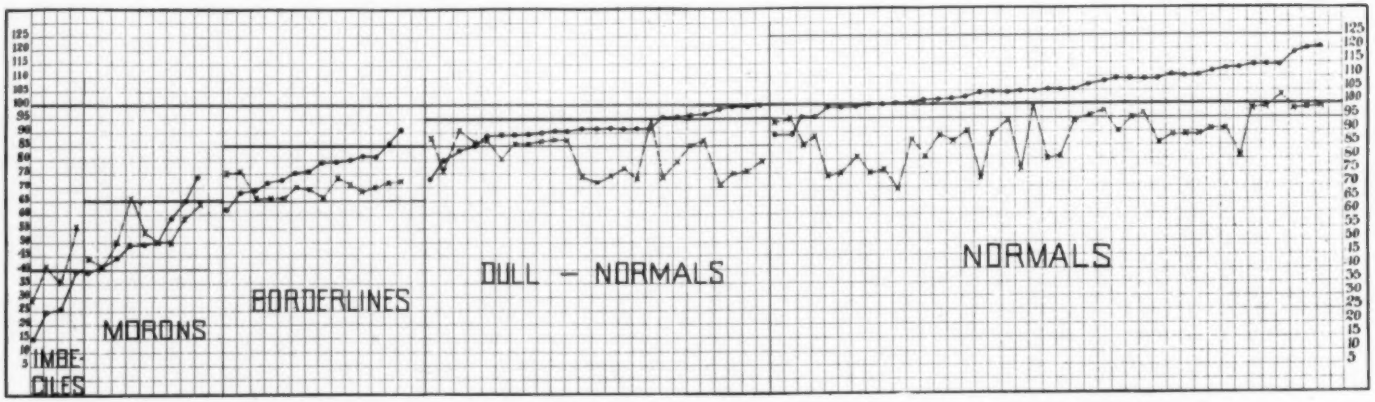

Chart 4.—Comparison of Binet Scale intelligence quotients arranged on the basis of Point Scale quotient.

Binet Scale. Table 1 shows the amount of "overlapping." The significant feature of this table is that by the Binet Scale, forty-two boys, who, we were satisfied, belonged to the normal group, had intelligence quotients below the group in which they were placed, whereas by the Point Scale eleven ranked above and only five below. Stated in another way, by the Binet tests, we should have had to place thirteen boys in the borderline group instead of in the dull normal group, whereas by the Point Scale only three such changes would have been necessary, if the results were to be rigidly interpreted by the intelligence quotient.

Let us emphasize again that the intelligence quotient, being based on group average norms in neglect of individual variations, involves a good deal of unavoidable error and to our mind should be used only for purposes of group study.

TABLE 1.—SHOWING COMPARATIVE AMOUNTS OF OVERLAPPING BY THE TWO SCALES

Cases	Binet Scale		Point Scale	
	Above	Below	Above	Below
Imbeciles, 15-40	2	0	0	0
Morons, 40-65	1	0	1	1
Borderlines, 65-85	0	0	2	1
Dull Normals, 85-95	0	13	8	3
Normals, 95	..	29	..	2
Total	3	42	11	7

Now it must be clear that one or the other of the scales is seriously faulty. That the Binet scale is at fault is suggested by the character of the discrepancies. In the first place, in unquestionably defective children the Point Scale gives a lower rating. Second, the discrepancies are least at the ages 6 to 10, when the Binet Scale has already been recognized as most accurate. Third, in the higher ages, when the Binet Scale has been found to be unsatisfactory, Point scores are obtained that correspond with normal expectation and in normal children increase with age (exception, at ages 14 to 15, when the score is stationary). Fourth, indications of exceptional intelligence are obtained by the Point Scale and not by the Binet Scale, which have been corroborated in a few of our cases by strong collateral evidence.

The Point Scale, moreover, appeals strongly to those who dislike the indirection of the Binet plan of scoring. To us the latter seems perfectly analogous to the recording of height, for instance, in terms of age. Besides being scientifically objectionable because indirect, it is also essentially inaccurate because it fails to take into account

the considerable variations of intelligence existing among normal individuals. Certainly but a small percentage of the many examinations that we have made during the last four years by the Binet Scale have shown an exact correspondence between mental "age" and chronological age.[6] The Point Scale score, on the other hand, is a direct statement of measurement and approaches the ideal of recording data in absolute rather than in relative terms.

We cannot leave the subject without mentioning a point which experience has brought frequently to our attention. This is the caution which is necessary in comparing individual scores with group average norms, as in the intelligence quotient, for the benefit of parents, guardians or teachers. We feel that in so doing the normal variations are frequently lost to sight and the stigma of retardation or pathologic deficiency wrongly fastened on the subject. It must be remembered that while the examiner can make allowances for normal variation, the laity will not do so. The figures for mental age are too readily compared with chronological age and insignificant degrees of variation require explanation which often serves only to cast suspicion still further. That this is by no means an unimportant or merely academic point is indicated by the strong public prejudice already created in many quarters against public school mental examinations. This is a question which must be promptly faced if mental examinations are to be generally introduced in American schools. The noncommittal (and more accurate) Point score has the inherent advantage that it can always be compared with *minimum* normal figures for age, instead of with a group average norm.

A word as to our method of deciding on the final diagnosis: We have not depended entirely on the score attained by either scale, but in a few instances have had to make allowance for various retarding environmental influences, such as deafness, faulty vision, and especially the effects of isolation and imperfect adaptation to surroundings consequent to foreign birth, foreign parentage and imperfect use of English. For these reasons, a few individuals have been provisionally placed in a category above that for which a strict interpretation of the tests would qualify them. In a few instances, also, we have modified the Point Scale diagnosis by a *favorable* discrepancy in the Binet Scale, but this has been necessary in but few cases.

PHYSICAL FINDINGS COMPARED WITH MENTAL STATUS

In Tables 2 and 3 we have arranged the more significant physical findings in columns parallel with our mental examinations. For this purpose we have taken ninety-three cases selected because of completeness, but on no other basis of exclusion.

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6. Yerkes, Bridges and Hardwick (p. 40) (Footnote 2) by a reconstruction of Goddard's data have brought out the same point very forcibly.

TABLE 2.—Summary of Physical—

	No.	Offense	Actual Age		Height		Height Percentile	Weight		Weight Percentile	Grip		Grip Percentile		Head		
			Yrs.	Mos.	In.	Mm.		Lbs.	Kg.		Right Kg.	Left Kg.	Right	Left	Girth, Cm.	Excess, Cm.	Deficit, Cm.
Normals	4	Stealing	17	1	66¼	1,680	43	138¾	63.0	66	41	42	30	47	55.5	....	....
	5	Stealing	12	3	61½	1,562	95	112	50.8	94	39	38	102	110	.....	....	....
	6	Incorrigible	15	4	66¼	1,680	79	138	62.5	85	46	46	87	91	56.9	2.2	....
	9	Dependent	15	11	64	1,625	57	133¼	60.4	88	39	41	65	83	58.5	3.5	....
	10	Dependent	10	9	55⅛	1,400	86	70½	31.8	74	19	22	70	93	53.5	0.7	....
	14	Stealing	16	2	60¾	1,545	10	105½	47.8	17	19	25	02	09	53.5	....	1.6
	15	Incorrigible	11	9	52⅝	1,337	27	69	31.3	41	20	22	60	85	51.5	....	1.6
	19	Stealing	15	5	67⅛	1,705	87	142½	64.5	91	38	32½	60	51	.....	....	....
	20	Incorrigible	16	3	64	1,630	34	131½	59.6	73	36	34	30	30	54	....	1.2
	26	Dependent	10	..	54⅞	1,395	84	79¼	35.8	91	16	21	30	92	52.2	....	0.7
	27	Stealing	13	7	55⅝	1,410	14	78½	35 5	22	23½	22½	43	48	53.6	....	....
	28	Truancy	15	6	63¾	1,622	55	122⅛	55.5	74	42	35	77	60	.....	....	....
	29	Stealing	11	5	55¼	1,405	70	72½	32.8	58	22	15	75	20	51	....	2.0
	35	Stealing	16	3	62¾	1,595	20	111½	50.5	29	....	....	....	....	.....	....	....
	37	Truancy	12	4	53⅝	1,362	20	83¾	37.9	71	24	22	70	70	54.3	1.0	....
	38	Dependent	12	5	59½	1,512	90	97½	44.2	91	29	23	91	75	52.5	....	0.8
	39	Dependent	12	8	58¼	1,480	80	96¾	43.8	91	31	27	93	91	55	1.7	....
	40	Dependent	13	7	61⅞	1,575	85	89¼	40.4	58	26	24	60	60	53.7	....	....
	41	Stealing	10	6	51⅝	1,312	36	70	31.7	73	20½	19½	83	83	52.8	....	....
	42	Burglary	16	8	60	1,525	32	96	43.5	08	35	33	25	27	52.3	....	3.1
	43	Truancy	12	5	55	1,400	39	92	41.7	86	25	28	75	95	54	0.7	....
	47	Dependent	13	..	54	1,378	08	74½	33.7	13	27	32	65	91	53.3	....	....
	57	Incorrigible	15	7	63⅞	1,625	57	119	53.9	68	30	31	30	45	54.5	....	0.3
	59	Stealing	14	..	57⅞	1,470	21	83	37.6	19	25	26	35	50	53.3	....	0.6
	62	Incorrigible	14	8	61	1,552	51	97½	44.2	50	30	29	60	65	53	....	1.3
	63	Burglary	15	7	62	1,575	35	134¼	61.0	88	41	40	73	81	54.3	....	0.4
	67	Incorrigible	14	2	61	1,552	51	112½	51.0	80	42	39	91	92	55	1.0	....
	69	Stealing	15	4	61⅛	1,555	27	107	48.5	41	39	44	65	88	51.3	....	3.4
	72	Truancy	8	7	47¾	1,215	39	52¼	23.7	37	16	15	77	77	49.5	....	2.7
	76	Truancy	10	7	51½	1,310	34	75¾	34.3	88	20	18	80	70	52.8	....	....
	79	Neglect	13	1	62¼	1,580	85	93¾	42.4	67	26	25	60	65	53.9	0.5	....
	82	Truancy	14	3	57¼	1,455	16	92	41.7	38	38	34	86	85	55	0.9	....
	83	Truancy	14	..	62¼	1,582	66	104	47.2	66	35	31	80	75	52	....	1.9
	87	Burglary	15	3	70¼	1,785	96	151¼	68.2	92	56	53	94	95	56	1.4	....
	91	Stealing	12	..	55⅞	1,420	50	73	33.1	32	24	23	70	75	51.5	....	1.7
	92	Stealing	16	4	67	1,705	70	125¼	56.8	57	34	38	20	50	53	....	2.2
	97	Incorrigible	9	8	53½	1,360	87	62½	28.3	62	21	21	91	93	50.6	....	2.0
	106	Stealing	13	9	58⅞	1,495	55	88¾	40.2	57	30	24	80	60	54	0.3	....
	107	Stealing	13	..	59¾	1,520	67	102	46.2	84	39	36	92	93	52	....	1.4
	108	Stealing	10	8	51¾	1,315	28	71¾	32.5	80	22	17	90	60	49.5	....	3.3
	110	Incorrigible	11	2	55⅝	1,415	75	83¾	38.0	90	24	23	85	90	51.5	....	1.4

Vision		Strabismus	Defective Hearing	Enlarged Tonsils	Teeth	Lymph Nodes		Prepuce	Testes	Skin Disease	Postural Defects	Point Scale Score	Binet Age	Normal = 100		Physical Percentile (Normal = 50)
O. D.	O. S.					Cervical	Epitrochlear							Binet I. Q.	Point Sc. I. Q.	
20/20	20/20	0	0	+	0	+	0	0	0	Fur.	0	96	15	88	100	26
20/20	20/20	0	0	0	0	0	0	0	0	0	0	88	12	98	119	98
20/20	20/20	0	0	0	0	0	0	R.	0	Acne	0	97	15.2	99	118	86
.....	.....	0	0	+	0	+	0	0	0	0	0	89	12.6	79	105	73
20/20	20/20	0	0	+	0	0	0	R.	0	Im.	0	67	10	93	106	81
20/20	20/20	0	0	0	0	+	0	0	0	0	0	93	15.4	95	108	10
20/20	20/20	0	0	0	D.	+++	0	Lg.	0	0	0	76	11.4	97	106	53
.....	.....	0	0	+	0	+	0	0	0	Im.	0	87	12.4	81	108	72
20/20	20/20	0	0	0	D.	0	0	0	0	0	0	85	12.4	71	99	42
20/20	20/20	0	0	0	D.	+	L. +	0	0	0	W.S.	68	10.4	96	113	74
20/20	20/20	0	0	+	D.E.	++	0	Lg.	0	0	W.S.	80	12.2	90	112	32
20/20	20/20	0	0	+	0	+	+	0	0	0	0	87	12.4	72	106	67
20/20	20/20	0	+	0	D.	++	0	Lg.	0	0	L.C.	79	11.2	98	114	56
20/20	20/20	0	0	0	D.	0	0	0	0	0	0	84	12	74	98	25
.....	.....	0	0	0	D.	+	0	Lg.	0	0	0	83	11	90	111	58
20/40	20/100	0	0	0	D.	0	0	0	0	Im.	0	72	10.6	86	96	87
.....	.....	0	0	0	0	0	0	0	0	0	0	78	11.4	89	104	89
20/20	20/20	0	0	0	D.E.	0	++	0	0	0	0	87	12	88	111	66
20/20	20/20	0	0	0	D.	++	0	R.	0	0	0	65	10.4	100	105	60
20/20	20/30	+C.	0	0	D.	0	0	0	0	Warts	W.S.	91	15	90	103	23
20/20	20/30	+C.	0	0	0	0	0	Lg.	0	0	0	82	12	97	109	74
20/100	20/50	0	0	0	0	++	0	R.	0	0	0	79	12.2	94	104	44
.....	.....	0	0	0	0	0	0	0	0	0	0	81	11.8	76	98	50
20/20	20/20	0	0	0	D.	0	0	Lg.	0	0	0	89	12.4	89	111	31
20/20	20/30	0	0	0	D.Ir.	++	0	R.	0	0	W.S.	80	12	82	100	57
20/20	20/20	0	0	0	0	0	0	R.	0	0	0	93	15	96	107	69
20/50	20/70	0	0	R.	0	+	0	R.	0	0	0	81	12.2	86	101	79
20/20	20/20	0	0	R.	0	++	0	0	0	0	0	80	12.4	87	99	55
20/30	20/30	0	0	R.	D.	0	0	R.	0	0	0	40	8.2	95	89	58
20/20	20/30	0	0	0	D.	0	0	0	0	0	0	70	10.6	100	113	68
20/30	20/30	0	0	++	D.Ir.	+	L. +	R.	0	0	0	78	11.4	89	103	84
20/20	20/20	0	0	0	0	+	0	0	0	0	0	82	11.6	81	102	56
20/20	20/20	+C.	0	++	D.Ir.	+	0	0	0	0	0	87	12.4	89	109	72
20/20	20/20	0	0	0	0	+	0	0	0	Fur.	St.	97	12.6	82	113	94
20/20	20/20	0	0	+	D.	+	0	0	0	0	0	71	10.6	88	96	57
20/20	20/20	0	0	0	0	0	0	0	0	0	0	91	12	74	104	49
20/40	20/50	0	0	0	D.	+	+	Lg.	0	0	0	51	9	93	89	84
20/20	20/20	0	0	R.	0	0	0	0	0	0	0	89	12.6	91	113	63
.....	.....	0	0	0	0	+	0	0	0	0	W.S.	83	11.8	91	109	84
.....	.....	0	0	0	0	+	+	0	0	0	0	68	10.4	97	108	65
20/20	20/20	0	0	+	D.	++	0	Lg.	0	0	W.S.	80	11.2	100	121	85

TABLE 2.—SUMMARY OF PHYSICAL—

	No.	Offense	Actual Age		Height		Height Percentile	Weight		Weight Percentile	Grip		Grip Percentile		Head		
			Yrs.	Mos.	In.	Mm.		Lbs.	Kg.		Right Kg.	Left Kg.	Right	Left	Girth, Cm.	Excess, Cm.	Deficit, Cm.
Dull Normals	1	Stealing	14	7	66	1,680	91	133¼	60.4	94	43	30½	92	73	56.5	2.2	....
	2	Stealing	14	5	56	1,425	09	87½	39.5	27	25	22	35	25	50.3	....	3.8
	8	Dependent	12	7	54⅞	1,395	36	71¾	32.5	27	23	19½	60	45	50	....	3.3
	16	Stealing	16	..	56	1,425	00	89¼	40.4	05	31	24	13	08	50	....	5.0
	17	Burglary	15	5	62⅛	1,580	37	114½	51.8	58	32½	36	41	63	53.3	....	1.4
	18	Incorrigible	10	7	54¾	1,392	83	80¾	36.6	91	25	24	93	96	52.5	....	0.3
	24	Truancy	11	4	57½	1,462	91	102¾	46.6	96	22	21	75	80	53.7	0.7	....
	25	Dependent	9	..	53¼	1,355	86	70¼	31.8	90	11	16	09	70	52.7	0.3	....
	33	Dependent	9	1	56⅛	1,430	95	81⅞	36.7	97	22	18	92	83	54	1.0	....
	36	Truancy	11	8	56⅛	1,427	81	75¾	34.3	70	23½	21	83	80	50.2	....	2.9
	46	Truancy	11	8	56½	1,437	84	70¾	32.5	54	19	20	50	70	52.5	....	0.6
	48	Dependent	10	..	54	1,372	76	74½	33.8	87	19	19	70	80	50.9	....	1.8
	54	Truancy	14	3	60¼	1,532	45	111½	50.5	78	40	35	90	87	56.9	2.9	....
	55	Dependent	14	3	60⅝	1,542	47	106	48.0	70	37	34	84	85	55	1.0	....
	56	Truancy	14	2	57	1,450	14	83¼	37.7	19	23	24	23	35	52	....	2.0
	71	Stealing	15	8	63⅜	1,615	50	129¾	58.8	84	44	43	82	87	56.3	1.5	....
	75	Dependent	16	8	63⅝	1,620	31	127¼	57.7	63	39	38	40	50	55.5	....	....
	77	Stealing	14	..	57⅛	1,452	15	88	39.9	29	30	28	60	60	52.7	....	1.2
	80	Truancy	15	1	62¾	1,595	42	115¾	52.4	61	35	30	50	40	54	....	0.5
	84	Truancy	13	4	54⅜	1,385	08	77¾	35.2	21	23	21	40	40	54	0.5	....
	90	Stealing	13	..	57⅝	1,465	41	88¾	40.2	57	24	23	45	50	53.3	....	....
	103	Stealing	13	8	62¼	1,585	86	128	58.0	94	28	31	70	90	53.5	....	....
	105	Dependent	13	6	61⅝	1,565	82	109¼	49.5	90	34	39	90	94	51.3	....	2.3
	109	Incorrigible	12	3	54½	1,385	32	87	39.4	76	21	17	40	20	52.3	....	0.9

—AND MENTAL FINDINGS—(*Continued*)

Vision		Strabismus	Defective Hearing	Enlarged Tonsils	Teeth	Lymph Nodes		Prepuce	Testes	Skin Disease	Postural Defects	Point Scale Score	Binet Age	Normal = 100		Physical Percentile (Normal = 50)
O. D.	O. S.					Cervical	Epitrochlear							Binet I. Q.	Point Sc. I. Q.	
20/20	20/20	0	+	R.	D.	+	0	R.	0	0	0	78	11	75	98	88
20/20	20/20	0	0	0	D.	+	+	0	0	0	0	64	11	76	80	24
20/20	20/20	0	0	0	D	+	0	Lg.	0	0	0	73	10	79	97	42
20/30	20/70	0	+	+	D.M.	0	0	R.	0	0	W.S.	84	12.2	76	99	07
20/30	20/200	+C.	0	R.	0	0	+	0	0	0	.....	82	12.2	79	100	50
20/20	20/20	0	0	0	-0	0	0	R.	Cry.	0	W.S.	53	9.2	87	85	91
20/30	20/40	0	0	0	0	+	0	0	Cry.	0	0	59	9.8	86	88	86
20/30	20/20	0	0	+	D.	+	0	Lg.	0	0	0	38	7.8	87	73	64
20/30	20/30	+C.	0	+	D.	++	L. +	Lg.	0	0	0	48	8.6	95	91	92
20/30	20/30	0	0	0	D.	+	0	0	0	0	0	59	10	85	83	79
.....	.....	0	+	R.	D.	++	L. +	R.	0	0	W.S. L.C.	63	10.6	91	88	65
20/20	20/30	+C.	0	0	D.	+	+	0	0	0	0	54	9.4	94	90	78
20/20	20/20	0	0	0	0	++	+	R.	0	0	0	74	10.4	73	92	75
20/20	20/30	0	0	++	D.	++	+	0	0	0	0	74	10.6	74	92	72
20/20	20/20	0	0	+	0	++	0	Lg.	0	0	0	70	11.2	79	88	23
20/200	20/20	0	0	0	D.	+	0	0	0	0	0	80	11.4	73	96	76
20/20	20/30	0	+	+	D	+	0	R.	0	0	0	80	12	72	91	46
20/30	20/30	0	0	+	0	+	++	0	0	0	W.S.	77	11	79	96	41
.....	.....	0	0	+	D.	++	0	0	0	0	0	79	10.6	70	98	48
20/20	20/20	0	0	+	D.	+	0	Lg.	0	0	0	71	11	83	92	27
20/20	20/20	0	0	0	0	0	0	R.	0	0	0	74	11	85	97	48
20/20	20/30	0	+	R.	Ir.	++	0	0	0	0	0	72	11.4	83	91	85
20/20	20/20	0	0	0	0	+	0	0	0	0	0	71	10.8	80	91	89
20/20	20/30	0	+	+	0	++	0	R.	0	0	0	66	10.6	86	89	42

TABLE 2.—SUMMARY OF PHYSICAL—

	No.	Offense	Actual Age		Height		Height Percentile	Weight		Weight Percentile	Grip		Grip Percentile		Head		
			Yrs.	Mos.	In.	Mm.		Lbs.	Kg.		Right Kg.	Left Kg.	Right	Left	Girth, Cm.	Excess, Cm.	Deficit, Cm.
Borderlines	11	Stealing	15	10	63⅞	1,625	57	119¼	54.1	69	31	30	35	40	55	....	....
	12	Stealing	14	2	58¼	1,477	24	96¾	43.8	49	31	35	65	88	55	1.0	....
	13	Dependent	12	..	55¾	1,420	49	76¾	34.8	48	20	18	30	30	50	....	3.2
	23	Truancy	15	..	60⅛	1,530	18	105¾	47.9	35	26	22	10	09	52	....	2.5
	31	Burglary	15	9	60½	1,540	21	119	53.9	68	32	29	40	35	.....	....	....
	34	Dependent	15	9	60¼	1,535	19	122¾	55.6	78	37	32	57	50	53	....	1.9
	49	Stealing	16	4	65	1,652	41	133	60.3	75	....	....	....	....	55.7	0.5	....
	50	Dependent	14	7	59½	1,512	47	110	49.8	76	32	26	70	50	54.5	0.3	....
	58	Stealing	12	5	49⅛	1,250	0	60¼	27.2	06	15	15	07	10	54	0.7	....
	70	Burglary	15	8	60⅞	1,550	25	117½	53.2	65	37	32	57	50	54	....	0.8
	74	Burglary	14	3	59¾	1,492	30	92½	41.8	39	30	28	60	60	56	....	2.0
	85	Incorrigible	14	2	53½	1,360	05	72¼	32.7	07	23	23	23	30	50	....	1.1
	86	Incorrigible	10	..	49¼	1,252	09	64¼	29.1	46	21	19	85	80	51	....	1.7
	94	Stealing	16	4	63¼	1,610	26	117	53.0	39	41	35	50	35	53	....	2.2
	95	Incorrigible	14	8	66¼	1,685	92	135½	61.4	95	37	28	84	60	53	....	1.4
Morons	7	Stealing	18	9	61⅞	1,575	—7	106¾	48.4	05	36	28	00	—8	56	0.3	....
	32	Stealing	17	..	65½	1,665	32	124	56.2	36	42	41	36	43	53	....	2.6
	45	Incorrigible	17	6	63¾	1,620	14	125½	56.8	38	24	29	03	08	51.5	....	4.1
	51	Incorrigible	12	10	53⅛	1,350	16	74¾	33.8	42	22	20	50	50	52	....	1.3
	52	Incorrigible	14	3	55	1,400	07	82½	37.4	18	21	19	10	10	50.5	....	3.5
	60	Truancy	17	2	62½	1,590	09	111	50.3	13	35	34	10	17	54	....	1.5
	73	Incorrigible	17	3	60⅞	1,550	08	108½	49.2	10	36	31	11	09	53	....	2.5
	93	Truancy	16	..	61⅜	1,560	12	126	57.2	59	38	36	37	40	57	....	2.0
	96	Truancy	13	9	52⅜	1,382	08	71¼	32.3	06	42	23	93	50	50.1	....	3.6
Imbeciles	3	Truancy	19	4	56⅞	1,445	—2	93½	42.3	—9	24	23	—26	—15	.....	....	....
	21	Incorrigible	16	3	65	1,652	41	143	64.8	87	41	44	50	77	55.2	....	....
	53	Incorrigible	8	5	46⅛	1,172	15	51¾	23.4	33	10	16	10	86	50	....	2.2
	101	Incorrigible	10	7	52⅝	1,337	55	87	39.4	93	11	7	07	—2	54.5	1.7	....

W.S. = Wing scapulae
E. = Defective enamel
Im. = Impetigo
Lg. = Elongated prepuce
Fur. = Furunculosis
L. = Left
D. = Decay
St. = Stoop shoulders
L.C. = Lateral curvature of spine

—AND MENTAL FINDINGS—(*Continued*)

Vision		Strabismus	Defective Hearing	Enlarged Tonsils	Teeth	Lymph Nodes		Prepuce	Testes	Skin Disease	Postural Defects	Point Scale Score	Binet Age	Normal = 100		Physical Percentile (Normal = 50)
O. D.	O. S.					Cervical	Epitrochlear							Binet I. Q.	Point Sc. I. Q.	
20/20	20/30	0	0	0	0	++	0	Lg.	0	0	0	64	11	70	75	50
20/20	20/20	0	0	0	D.	0	0	0	0	0	0	55	9.6	68	69	57
20/30	20/30	C. +	0	+	D.	+	0	Ph.	Cry.	0	0	46	9	75	62	39
20/20	20/20	0	+	++	D.	+	0	0	Cry.	0	W.S.	65	10.6	71	81	18
20/20	20/20	0	0	R.	D.	+	+	R.	0	0	L.C.	60	10.6	67	72	41
20/30	20/40	0	+	++	D.	+	0	R.	0	0	L.D.	64	10.4	66	77	51
20/200	20/50	0	0	0	D.	0	0	0	0	0	0	78	11.8	72	91	
20/20	20/20	0	0	+	0	+	0	0	0	0	St.	59	9.8	67	73	61
20/50	20/40	0	0	R.	0	+	+	Lg.	0	0	0	51	9.4	76	68	06
20/20	20/20	0	0	+	D.	+	0	0	0	0	0	64	10.8	69	76	49
20/20	20/30	0	0	+	D.	+	+	Ph.	0	0	0	62	10.4	73	78	47
20/20	20/20	0	0	R.	D.	0	0	0	0	0	0	69	10.2	72	86	16
20/30	20/30	0	0	0	D.	++	0	0	0	0	0	51	8.6	86	85	55
20/20	20/20	0	0	0	0	++	0	0	0	0	0	70	11	68	81	38
20/20	20/40	0	0	++	0	++	0	Lg.	0	0	0	64	10.4	71	80	83
20/30	20/100	0	0	0	D.	0	+	0	0	0	St.	55	9.4	50	58	—3
20/30	20/30	0	+	0	D.	0	0	0	0	Im.	St.	35	7.4	44	39	37
20/40	20/40	0	0	0	D.	0	0	0	0	Fur.	0	47	8.8	50	50	16
20/20	20/20	0	0	+	0	+	0	R.	0	0	0	36	8.4	66	47	40
20/40	20/30	0	0	0	0	+	0	R.	0	0	0	35	8	56	44	11
20/40	20/40	0	0	0	0	0	0	R.	0	0	W.S.	38	7.2	42	42	12
20/100	20/100	+C.	+	0	0	0	0	0	0	0	0	59	10	58	65	10
20/30	20/70	0	++	+	0	0	0	0	0	Acne	0	63	10.2	64	74	37
20/30	20/30	0	+	0	0	+	0	0	0	0	0	39	8	58	49	40
20/20	20/30	0	+	0	0	0	0	0	0	0	St.L.C.	15	6.4	33	16	13
20/40	20/50	+D.	0	0	D.	0	0	0	0	0	0	22	6.6	40	26	64
.....	.....	0	0	+	D.	+	0	Lg.	0	0	W.C.	17	5.4	64	39	36
.....	.....	0	0	0	D.	++	0	0	0	0	0	15	4.4	24	24	38

C. = Convergent
Cry. = Cryptorchidism
Ld. = Lordosis
D. = Divergent
M. = Malocclusion
R. = Removed
Ir. = Irregular
Ph. = Phimosis

TABLE 3.—SUMMARY OF PHYSICAL EXAMINATIONS

	Cases						
	Normal	Dull Normal	Borderline	Moron	Imbecile	Total Normal	Total Defective*
Number of cases	41	24	15	9	4	65	28
Average height percentile	52.7	52.6	30.8	11.0	27.3	52.7	23.1
Average weight percentile	52.7	63.7	53.0	25.4	51.0	56.7	43.9
Average right grip percentile	66.3	59.4	48.1	27.8	10.3	62.7	34.4
Average left grip percentile	70.5	63.0	44.8	24.3	36.5	66.6	35.4
General percentile average	60.6	59.7	44.2	22.1	31.3	59.7	34.2
Minimum height percentile	8	0	0	—7	—2		
Maximum height percentile	96	95	92	32	55		
Variation	88	95	92	39	57		
Minimum weight percentile	8	5	6	5	—9		
Maximum weight percentile	94	97	95	59	93		
Variation	86	92	89	54	102		
Minimum right grip percentile	2	9	7	0	—26		
Maximum right grip percentile	102	93	85	93	50		
Variation	100	84	78	93	76		
Minimum left grip percentile	9	8	9	—8	—15		
Maximum left grip percentile	110	96	88	50	86		
Variation	101	88	79	58	101		
Head girth deficit	0.5	0.7	1.0	2.7	0.2	0.6	1.4
Percentage cases with							
Vision below 20/30, O. D.	11.8	4.5	13.3	45.4	50.0	8.9	26.9
Vision below 20/30, O. S.	11.8	13.6	26.7	45.4	50.0	12.5	34.6
Strabismus	7.3	12.5	6.7	11.1	25.0	9.2	10.7
Defective hearing	2.4	25.0	13.3	45.4	25.0	10.8	25.0
Hypertrophied tonsils	24.4	41.7	46.6	22.2	25.0	30.8	35.7
Tonsils removed	9.8	12.5	20.0	0	0	10.8	10.7
Tonsils removed or hypertrophied	34.2	54.2	66.7	22.2	25.0	41.6	46.4
Dental defects	48.8	62.5	66.7	33.3	75.0	53.8	57.2
Enlarged cervical glands	58.6	83.3	75.0	33.3	50.0	67.8	60.8
Enlarged epitrochlear glands	14.7	33.3	20.0	11.1	0	21.6	25.0
Skin disease	17.1	0	0	33.3	0	10.8	10.7
Postural defects	19.5	16.7	26.7	33.3	50.0	18.5	31.2

* Including borderlines.

The physical percentile (general average) was obtained by averaging the percentiles for height, weight and right and left grip and therefore is comparable with the "psychophysical" percentile of Doll,[7] except that he included also the figure for vital capacity. The reader is again reminded that the norm for the intelligence quotient is 1.00, and for all percentiles is 50. The averages in the small imbecile group are unduly influenced by a single physically exceptional case.

The chief feature of our results, that the physical group average is proportional to the mental status of the group, is in agreement with the work of Porter, Christopher, Smedley, Doll and many others (see Doll's monograph[7]), but we should like to emphasize again, by reference to our figures, that this is true of groups only. For instance, nineteen of our forty-one normal cases (46.4 per cent.) showed height percentiles below 50. On the other hand, one imbecile and three borderlines (14.6 per cent.) out of the defective groups were above 50 for height. Seventeen of the dull normal and normal groups (26.2 per cent.) were below 50 for weight, while ten (35.7 per cent.) of the defectives were above 50. Eighteen (27.8 per cent.) of the normal groups were below 50 for right grip, and fourteen (21.6 per cent.) for left grip, while of the defective eleven (39.3 per cent.) were above 50 for right grip and twelve (42.8 per cent.) for left grip. These facts do not affect the fact of physical inferiority in mental defectives as a *group*, but it certainly rules out any diagnostic value in physical measurements for mental capacity.

Certain special phases of the physical examination could be related to mental status. In the dull normal group it will be seen that the percentage of boys with hypertrophied tonsils and chronic lymphadenitis (probably also with adenoid growths) was much larger than in the normal group, and this fact may be of considerable significance in explaining retardation.

Faulty vision[8] was found more frequently in the retarded and defective groups than in normal groups. This confirms Burpitt's[9] observations. Only 8.9 per cent. of our normals had less than 20/30 vision for the right eye, and 12.5 per cent. for the left, while 26.9 per cent. of the defectives had less than 20/30 for the right, and 34.6 per cent. for the left. The differences between the normal and dull normal groups for vision are not striking. Defective hearing, on the other

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7. Doll, E. A.: Anthropometry as an Aid to Mental Diagnosis. Publications of the Training School at Vineland, N. J. Research Dept. No. 8, February, 1916.

8. For summaries of previous work on the special senses in defectives the work of Whipple (Manual of Mental and Physical Tests, Baltimore, Warwick and York, 1914, Part 1) should be consulted.

9. Burpitt, H. R.: Mental Retardation, Nutrition and Eyesight in Schoolchildren. Ophthalmoscope, 1915, **13**, 442.

hand, was found in 25 per cent. of the dull normals as against 2.4 per cent. for the normals, a highly suggestive fact. For the normal group as a whole 10.8 per cent. showed defective hearing, as compared with 25 per cent. for the defectives.

Postural defects were also more frequent in the defectives, occurring in 31.2 per cent. and in only 18.5 per cent. of these normals. This observation has, we believe, not been made before.

Other physical abnormalities, such as dental caries, cervical and epitrochlear adenitis, genital defects and skin disease, are about evenly distributed between the normal and defective groups.

OFFENSES

In a group of eighty-five boys not committed for dependency or neglect, the offenses were distributed as shown in Table 4.

TABLE 4.—DISTRIBUTION OF OFFENSES

	Stealing	Truancy	Incorrigibility	Burglary
Normals	20	5	7	4
Dull normals	7	9	5	0
Borderlines	6	2	2	4
Morons and imbeciles	3	4	7	0

Stealing and burglary formed 54.5 per cent. of the offenses in the normal groups and 46.4 per cent. in the defective groups, not a striking difference.

HOME CONDITIONS

In the case of defectives, feeblemindedness is in itself a sufficient explanation of delinquency. In the case of normal children, however, it was believed that a study of environment might have considerable sociologic interest. Such an investigation was carried out by Miss Rosenshine in the case of fifty-five of our normal or dull normal groups, and is summarized in Chart 5. We do not care to make a detailed analysis of these findings, leaving this task to more experienced interpreters of social conditions, but we may point out that in nearly every case the home conditions were very unfavorable; this and the lack of home influence of any kind are important contributing causes of juvenile delinquency.

A frequent problem with which we have met is the case of the children of immigrants, especially of the non-English speaking class. It frequently happens that the parents have failed to adapt themselves to their new environment and their children suffer the double handicap of poverty and the conflict, expressed in many ways, between old and

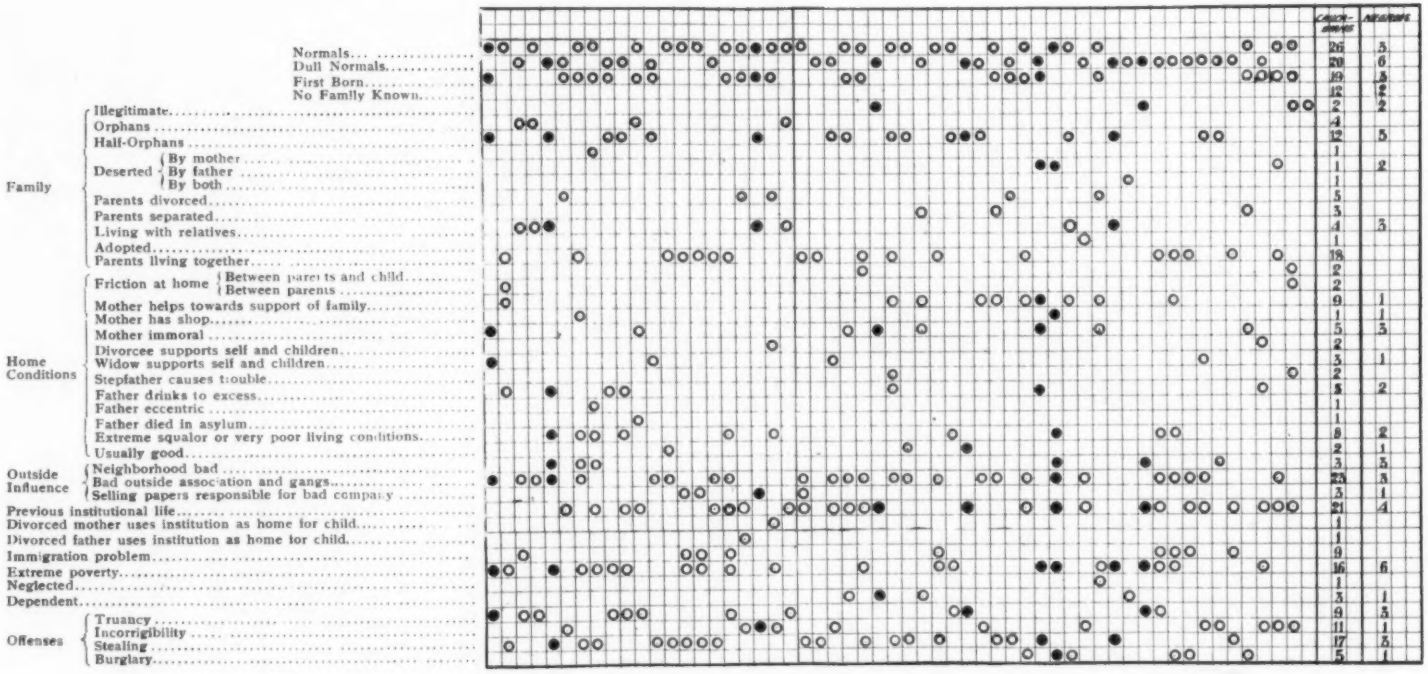

Chart 5.—Summary of environmental conditions, normal and dull-normal groups. Whites = ○; negroes = ●.

new world modes of life. This means in nearly every case the loss of home influence.

The frequency of illegitimacy, prostitution, divorce, and heavy drinking in the families investigated also deserves mention.

CONCLUSIONS

Our survey is of a group of juvenile offenders, and so is of importance as reemphasizing the physical, mental and moral problems which the community must solve before it can hope to deal helpfully with the problem of delinquency. The physical findings are, on the whole, those of neglected hygiene, secondary in most cases to poverty. The mental findings show that of the boys studied 43.6 per cent. were normal, 25.5 per cent. dull normal (total normals 69.1 per cent.), 17 per cent. borderline, 9.6 per cent. morons, and 4.3 per cent. imbeciles (total defectives 30.9 per cent.). A study of the normal and dull normal groups brings to light various unfavorable home conditions. We cannot avoid the conclusion that for the normal boys improved environment, improved personal hygiene, and better general education would have made useful citizenship possible.

In any group such as the one with which we have dealt, the first indication is obviously to separate the normals and dull normals from the rest, to keep the borderline cases under observation, with careful and systematic attempts at education in order to determine and develop latent ability; and finally to segregate promptly those who are beyond question feebleminded.

Of these problems the reeducation of normal individuals who have become delinquent is at once the most difficult, the most hopeful and the most important. Without special and extremely capable training these boys, who have potentialities not only of "floating" in society and of becoming good citizens, but even, in some instances, of attaining positions of superiority, will inevitably return to a career of delinquency or crime from which at a slightly later age it will be impossible to rescue them. Any institution such as the one we have studied must choose whether it will be a place of intelligent rehabilitation or a feeder to the criminal class.

About half the physical examinations were made by Dr. H. H. Yerington and Dr. W. L. Adams, and several special eye examinations were performed by Dr. Hans Barkan. For these, grateful acknowledgement is made.

THE CAUSE OF SUDDEN DEATH IN STATUS LYMPHATICUS *

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Status lymphaticus is a constitutional hereditary anomaly characterized anatomically by certain external peculiarities of configuration, by hypoplasia of the cardiovascular apparatus, by hyperplasia of the thymus gland and of the lymphoid tissues in other localities, and, incidentally, by congenital structural defects in different viscera. Clinically, the condition is not infrequently terminated by sudden death on apparently trivial provocation, oftenest in children, but occasionally in adults.

Among the first to recognize the anatomic defects in status lymphaticus and to apply their visible manifestations to purposes of diagnosis during life were Dr. Charles Norris in New York and Neusser in Vienna. Clinically, the condition may be identified in the living male subject by bodily configuration simulating that of the female. The face is beardless, or nearly so, and the axillary and other hairs are scanty, the skin is smooth and unusually delicate, the pubic hairs are sharply defined in a transverse direction, the waist is narrow, and the thighs are gracefully arched. In the female the diagnosis during life is rather more difficult, and rests largely on features which are but an accentuation of those normally encountered, namely, the thin, delicate skin, the narrow waist and the arched thighs, together with the small axillary fat pads and the scanty growth of hair on them.

The anatomic changes in the deeper parts are of practical significance. Among the first 4,000 necropsies at Bellevue Hospital status lymphaticus was encountered in 249 subjects, or in 6.2 per cent.; 197 times in males and 52 times in females. In our necropsy protocols we are accustomed to describe two types — status lymphaticus and status lymphaticus recessivus. In the Bellevue Hospital cases, 118 examples of status lymphaticus were encountered, and 89 cases belonging to the recessive type. In 42 instances border line cases were observed.

The anatomic changes were distributed thus:

Status Lymphaticus. — Of 118 cases of straightforward status lymphaticus the thymus was enlarged in every case. The weight of

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* From the Pathological Laboratories of Bellevue and Allied Hospitals; Director, Dr. Charles Norris.

the thymus was recorded 66 times. The youngest subject was a child of 8 hours whose thymus weighed 70 gm. The oldest subject was 38 years of age and the thymus likewise weighed 70 gm.

The average weight of the thymus, grouped according to years, was as shown in the table.

TABLE OF AVERAGE WEIGHTS IN STATUS LYMPHATICUS

Age, Years	Average Weight, Gm.	No. of Cases
Under 1	25.0	13
1- 5	18.0	14
6-10	24.0	5
11-25	22.0	5
16-20	23.0	9
21-30	27.8	17
31-40	33.8	3

Of the 118 cases, the faucial tonsils were enlarged 61 times (51.7 per cent); the lingual tonsils 58 times (49. per cent); the pharyngeal lymphoid follicles 45 times (38 per cent.). Peyer's patches and the solitary follicles were each enlarged 105 times (89 per cent.). The axillary nodes were enlarged 11 times (9 per cent.); the inguinal nodes 12 times (10 per cent.), and the cervical nodes 15 times (12.8 per cent.).

The spleen was small or normal in size in 68 cases (70 per cent.); the Malpighian follicles were hyperplastic 101 times, or in 85 per cent.

Status Lymphaticus Recessivus.—This is attended by retrogression of the lymphoid tissues, the other anatomic anomalies remaining, of course, unchanged. The recessive condition is encountered oftenest in individuals who have passed the twentieth year. Rarely is it seen before that age. It is attended by recessive changes in the lymphoid follicles varying in both location and extent, the most frequent indication of regression being found in the follicles of the intestinal tract, where atrophic changes occur in inverse proportion to the hyperplastic alterations in status lymphaticus, and in the thymus gland, which, in well marked instances, is practically completely replaced by fat. In the case of the lymphoid follicles of the intestine, lymph nodes and spleen the recessive changes are ascribable partly to natural involution and partly to connective tissue replacement of the germinal follicles, the latter process striking at the source where lymphoid cells are regenerated, while in the case of the thymus, recession is almost entirely an involutional change.

Of 89 cases of recessive status lymphaticus the thymus was weighable 19 times, and the average weight was 18 gm. In the remaining 70 cases (78.6 per cent.) the thymus was completely replaced by fat.

Of the 89 cases of recessive status lymphaticus the faucial tonsils were hyperplastic 25 times (28 per cent.); the lingual tonsils 32 times (36 per cent.), and the pharyngeal tonsils 19 times (21 per cent.).

Peyer's patches were atrophied 61 times (68.5 per cent.) and the solitary follicles 37 times (41 per cent.). The splenic follicles were enlarged 37 times (41 per cent.) and atrophied 39 times (43.9 per cent.). The spleen was normal or small in size in 52 cases (71 per cent.).

Of the 249 cases of status lymphaticus the heart was small 127 times (51 per cent.), and in 101 cases (40.5 per cent.) the aorta was diminished in caliber, 71 times by actual measurement. In 78 cases (31 per cent.) the elasticity of the aorta was increased, and in 66 cases (26.5 per cent.) the vessel was described as unusually thin.

The practical application of the anatomic changes in status lymphaticus may be described under two heads:

Anatomic Changes.—The external configuration of the body is of great importance, since it serves at once to identify individuals, especially children, who are to be watched with the utmost care in order that they may be safely guided through events which, ordinarily, are free or relatively free from danger. To them the trivialities of life are apt to assume the magnitude of perils — vaccination, the injection of antitoxin, thoracentesis, light anesthesia, submersion—any one of these or their like may eventuate in sudden death. Moreover, children of this type are not only unusually susceptible of infection, but they bear infection badly, a clinical fact which was long ago recognized by the elder Gross when he described the "angelic child"—fair haired or dark, blue eyed or brown, thin lipped, pink cheeked, alert to the point of precocity, with velvety skin, thin, long bones, shapely limbs and graceful movements — born beautiful, but a prey to infection and a bad risk.

In adult life status lymphaticus is of interest from a sociologic as well as from a medical standpoint. Thus it is of striking frequency among individuals of unstable emotional qualities, including alcohol and other drug habitués (Emerson), criminals and suicides (Norris, Schultze, Symmers, Bartels), neurasthenics and savages (Symmers), epileptics and the insane (Karpas), and subjects of such glandular perversions as Addison's disease, exophthalmic goiter, acromegalic giantism and other forms of pituitary disease.

Sudden Death.—Sudden death in subjects of status lymphaticus has often been ascribed to pressure of the enlarged thymus on the trachea, but those who have most carefully studied the condition postmortem do not hold to this view; in fact, the theory is now credited in few quarters. At Bellevue Hospital we have never been able to implicate the enlarged thymus as a mechanical factor in the production of death. On the contrary, sudden death in status lymphaticus is an exceedingly subtle procedure, and is most logically explicable, I believe, by the laws

of anaphylaxis as promulgated by the experimental pathologists and exemplified by certain types of disease in man. This opinion is based on anatomic and histologic observations. Thus, the lymphoid tissues are most abundantly developed in youth and early adolescence, and undergo diminution as age advances — an observation which assumes importance in view of the fact that in status lymphaticus the danger of sudden death is greatest during that period of life in which the lymphoid tissues are most flourishing, and is practically unknown in the recessive stage. It is likewise significant that, in status lymphaticus, those lymphoid tissues which display the highest degree of hyperplasia are to be found in the mucous membrane of the gastro-intestinal tract where, of all the lymphoid structures, they are the ones most actively engaged in the process of filtration, in the course of which they are constantly subjected to the destructive action of a great variety of toxic substances, both chemical and bacterial. It is important to note, therefore, that, in children suddenly dead with all the physical attributes of status lymphaticus, microscopic examination of the lymphoid tissues throughout the body reveals constant and characteristic changes in the germinal follicles in the form of necrotic lesions marked by excessive disintegration of nuclei and the formation of "nuclear dust." These changes may be demonstrated in myriads of follicles; in fact, it is rarely possible in these circumstances to find a follicle that is intact. In other words, the histologic changes in question tend to show that sudden death in status lymphaticus is in some way connected with the release of nucleoproteins formed as a result of destruction of innumerable germinal follicles. It is scarcely conceivable, however, that death is directly traceable to the sudden and simultaneous destruction of a large number of follicles, since the interval between injury and death is far too short, in the majority of cases, to permit so many necrotic lesions to occur throughout such a wide distribution. It is more probable, I think, that death is of the nature of an anaphylactic phenomenon, sensitization being expressed in structural terms by the necrotic germinal follicles and, chemically, by the release of nucleo-proteins which, although not strictly foreign, are none the less pathologic, and comparable, in a toxicologic sense, to alien products. Previous to the expiration of the so-called anaphylactic incubation period the lymph nodes are subjected to the action of destructive substances which serve to bring about still further disintegration of germinal nuclei, thus providing the requisite quantity of specific protein to complete the anaphylactic reaction. The destructive substances in question may be introduced in the form of antitoxins hypodermically injected, or as vaccines applied by scarification or otherwise, or as substances derived from intestinal absorption, or manufactured in the

process of shock induced even by such simple procedures as the prick of a needle, sudden exposure to cold, and a number of similar events. That apparently trivial and negligible affairs are sometimes attended by extraordinary physical or chemical revulsions is a recognized fact in medicine, and is illustrated, among other things, by the almost instantaneous jaundice which sometimes is occasioned by severe fright, so that the occurrence of equally remarkable reactions in status lymphaticus is by no means unprecedented.

That acute necroses may occur in irregular showers is shown by the presence of all stages of both the necrotic and reparative processes in the same lymph node and in different nodes of the same body. This fact is of moment, in that the anaphylactic reactivity of the body is determined by the number of acute necrotic lesions in the germinal follicles, or, in other words, the anaphylactic incubation period in man, as in experimental animals, varies with the initial dose of the sensitizing protein. For example, in guinea-pigs injected with small amounts of horse serum, from twelve to fourteen days suffice for sensitization. With larger doses, however, the interval may be extended to weeks or even months. In subjects of status lymphaticus it is naturally to be expected that early sensitization follows a small shower of necroses and that larger showers are succeeded by a longer period of incubation, the reactive ranges thus varying within wide limits. At one moment the tissues are exquisitely tuned and await only the receipt of a sufficient quantity of specific protein to react fatally, while at another moment events are so timed that the same quantity of specific protein exerts no such effect, a fact which tends to explain why certain subjects of status lymphaticus survive procedures which, in others, are attended by disaster. In this instance it is apparently a question of anaphylactic reactivity dependent on the number of acute necrotic lesions in the lymph nodes and on the interval that has elapsed since their inception. In other circumstances, however, immunity from sudden death in status lymphaticus seems to be due to the process of so-called anti-anaphylaxis, which may be produced artificially in animals by injecting them, before the expiration of the anaphylactic incubation period, with a single large dose of the same foreign protein which was used for purposes of sensitization. The animal recovering from the symptoms induced by the second injection is thereafter immune to the effects of the same protein. Precisely the same process seems to occur in status lymphaticus when, following sensitization to a specific protein and before the anaphylactic incubation period is over, the same protein is formed in larger quantities following a shower of necroses in the germinal follicles. After this the individual is immune and is enabled to withstand not only trivial

shocks, but even perils of great magnitude, such as infections, major accidents and operations demanding deep anesthesia. This interpretation is borne out by the fact that, in subjects of status lymphaticus, especially adults, in whom death is attributable to causes other than anaphylaxis, acute necrotic changes in the germinal follicles of the lymph nodes are exceedingly rare, or may not be demonstrable at all, the vast majority of the follicles exhibiting, on the contrary, definite signs of connective tissue replacement, showing that necrotic lesions have been present at some time, but have been repaired.

It is quite possible, on the other hand, that in certain cases of status lymphaticus the phenomenon of allergy obtains; that is to say, the patient is hypersensitive to the effects of a second dose of the specific protein with which he has been sensitized, in which event one shower of follicular necroses following rapidly on another might readily give rise to a fatal reaction, or, failing this, to certain of the lesser phenomena to which subjects of status lymphaticus are liable, and which vary in intensity from simple urticarial rashes to convulsive disorders. It is along this line of procedure, I think, that we must seek to obtain a comparatively harmless diagnostic test which will indicate to what extent, if any, a given patient is susceptible to the effects of nucleoproteins derived from the destruction of lymphoid cells.

Finally, it is significant that, in guinea-pigs, death from anaphylaxis is accompanied by asphyxia due to tetanic spasm of the muscles in the smaller bronchi, and similar changes in human beings are undoubtedly responsible for such respiratory phenomena as asthmatic attacks, etc. Sudden death in status lymphaticus is likewise a respiratory event, directly traceable, however, not to peripheral causes, but most likely, to disturbances of the respiratory center in an individual whose nervous, vascular and lymphatic systems are notoriously unstable.

A second and less subtle cause of sudden death in status lymphaticus sometimes occurs in young adults as a direct result of rupture of a peripheral or central cerebral artery spontaneously, or on apparently trivial traumatism. A number of cases of this description have been observed in the past ten years in the necropsy room at Bellevue Hospital. Many of them were of profound medicolegal importance. In this type of case the subject always presents the bodily configuration and the other anatomic features of status lymphaticus, and necropsy reveals unusually delicate cerebral vessels, the deficiency being most noticeable in the muscular coat, rupture occurring apparently under the influence of excitement, exertion or other cause of sudden elevation of blood pressure. For example, in a case recently investigated postmortem by Dr. O. H. Schultze, the subject died suddenly while engaged in a fisticuff, and death was found to be due to spontaneous rupture

of an hypoplastic meningeal vessel, the party of the opposite being thus saved from a probable conviction on the charge of manslaughter.

CONCLUSIONS

Sudden death in status lymphaticus may be brought about in at least two different ways. The first and most frequent cause is of the nature of an anaphylactic reaction due to sensitization of the body by a specific nucleoprotein formed in the lymph nodes as the result of necrosis of numbers of germinal follicles. Before the so-called anaphylactic incubation period has expired the tissues are again subjected to the action of the same protein formed in the same type of tissue in response to an apparently trivial injury, and, in this way, the anaphylactic reaction is completed.

A second cause of sudden death in status lymphaticus is to be found in the form of spontaneous rupture of an hypoplastic cerebral vessel, or rupture following apparently trivial injury, the deficiency in the vessel wall being most noticeable in the muscular coat.

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THE TREATMENT OF SECONDARY ANEMIA IN INFANTS BY BLOOD TRANSFUSION*

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The cases covered in this report were referred to me because of simple secondary anemia or because of anemia and malnutrition. Various food combinations had been tried and all patients had received medical treatment for the anemia.

TECHNIC OF BLOOD TRANSFUSIONS

The transfusions were made at the Babies' Hospital by Dr. E. A. Morgan and Dr. T. C. Clark, former resident physicians of the hospital.

The blood of the donor was proved fit by the absence of agglutination and hemolysis.

The following instruments were used: Five record syringes, Lindemann needle, exploratory needle, hypodermic syringe with needle, scalpel, clamps, catgut, silk and needles.

The following technic was employed: The skin over the median basilic vein was cleaned and cocainized. An incision was made and about 2 cm. of the vein exposed and dissected free from the surrounding tissues. A small opening was made into one side of the vein with a pair of scissors, and a Lindemann needle inserted. The vein below the needle was then tied off and another suture was placed over the vein to hold the needle in place. A small amount of salt solution was introduced, to make sure that there was no leakage.

A rubber tourniquet was placed on the arm of the donor and the skin cleansed over the most prominent vein. The exploratory needle was inserted directly into the vein and the blood drawn with a record syringe until it was full. This syringe was then handed to the operator working on the child and the blood inserted into the child's vein. At the same time a fresh syringeful of blood was being obtained from the donor. Each syringe was well washed out with sterile salt solution before being used again to collect blood. This procedure was continued until the required amount had been transfused.

The immediate and later results are shown in the accompanying table.

The results in all but one patient were satisfactory. The patient in Case 4 was transfused twice, and in each instance there was improvement as shown by the blood examination, but it failed to hold longer than a few weeks. This child made a gain in weight, but the general improvement was not satisfactory. The abdomen was greatly distended, not unlike Hirschsprung's disease. In the other cases there was no return of the anemia and subsequent growth and development was all that could be hoped for.

Cases 5 and 7 require special mention. In addition to the anemia there was extreme malnutrition. The patient (Case 5) was very weak and exhausted to the point of development of small petechial hemor-

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THE BLOOD IN SECONDARY ANEMIA BEFORE AND AFTER TRANSFUSION

Sex	Age, Months	Weight, Pounds	Blood Before Transfusion		Amount Transfused, C.c.	Blood After Transfusion						Weight, Pounds
			Hemoglobin, per Cent.	Red Blood Corpuscles		Hours After	Hemoglobin, per Cent.	Red Blood Corpuscles	Months After	Hemoglobin, per Cent.	Red Blood Corpuscles	
F.	12	12.5	14 (Sahli)	2,400,000	175	24	45 (Sahli)	5,120,000	17	70 (Fleischl)	4,600,000	26.8
F	18	24.0	37 (Sahli)	3,900,000	200	24	55 (Sahli)	5,760,000	14	90 (Fleischl)	5,000,000	32.0
M	12	16.4	32 (Sahli)	4,480,000	300	24	78 (Sahli)	5,150,000	3	60	3,840,000	19.5
									6	52	4,400,000	22.2
									12	68	5,000,000	25.5
									18	65 (Dare)	5,000,000	28.0
F.	23	15.10	20	2,800,000	140	24	55	3,100,000	1	37	2,700,000	16.7
	24	16.7	30 (Sahli)	.....	200	24	90 (Sahli)	4,320,000	7	.. (Dare)	.....	19.8
F.	6	11.0	37 (Sahli)	1,600,000	150	24	85 (Sahli)	4,000,000	7	65 (Dare)	4,800,000	19.5
M.	10	12.5	33 (Dare)	4,300,000	200	24	58 (Dare)	4,900,000	1	58 (Dare)	5,000,000	12.8*
F	12	12.4	35 (Sahli)	3,120,000	170	24	62 (Sahli)	4,040,000	1	50	5,320,000	14.0
									3	50	4,140,000	17.4
									6	56 (Dare)	4,400,000	19.8
M	16	21.0	35 (Sahli)	3,310,000	200	24	55 (Sahli)	3,400,000	1	60 (Dare)	4,500,000	24.6
									6	55 (Fleischl)	4,100,000	25.12

* Died of acute meningitis three months later.

rhages over the body, particularly over the lower abdomen. Several weeks of treatment were required to bring her to a reasonably normal condition. In Case 7 the patient was a very anemic twin and failed to grow regardless of the food given. She made a prompt and rapid response to transfusion.

The table shows the weight increase and the blood findings before and after transfusion, but it cannot record the marked change in the patients—the change from sickly, whiny infants into happy, apparently well infants. The patients were transformed from those with a digestive capacity barely able to maintain existence into those that took on the normal constructive processes of early life.

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Poliomyelitis, Acute Anterior.—M. D. Morrison.
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Poliomyelitis, Anterior, Diagnosis of.—C. Miller.
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Poliomyelitis, Anterior, Mechanical and Surgical Treatment of.—A. J. Gillette and C. C. Chatterton.
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Poliomyelitis, Epidemic, or Infantile Paralysis, Epidemiology of.—A. G. Nicholls.
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Poliomyelitis, Reflexes in, Condition of.—J. G. Regan.
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Typhoid Outbreak, Milk-Borne, in Children.—S. T. Lipsitz.
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TUBERCULOSIS AND SYPHILIS

Perthes' Disease, Etiology of.—Percy Willard Roberts.
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Syphilis, Hereditary.—W. L. Kendall.
Oklahoma State Med. Assn. Jour., 1917, **10,** 387.

Teeth, Molar, Fifth Cusp on, Not Necessarily Sign of Syphilis.—M. Mozer and C. Chenet.
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Tuberculosis, Papillary Cutaneous Reaction in.—H. A. Ellis.
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GASTRO-INTESTINAL SYSTEM

Appendicitis in Infants.—I. A. Abt.
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Constipation, Habitual, V. T. E. H. Thaysen.
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Helminths in Stools, to Facilitate Search for.—J. Carles and E. Barthélemy.
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Hernia, Strangulated, in Child Three Weeks Old.—J. H. Ingham.
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Hirschsprung's Disease. Case of Megacolon: Megasigmoid.—W. R. Jackson.
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Megacolon and Megarectum, Congenital.—L. Bard.
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Pyloric Stenosis, Hyperplastic.—D. W. Palmer.
Ann. Surg., 1917, **66,** 428.

Worms, North American Parasitic, Structure and Classification of.—H. B. Ward.
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RESPIRATORY SYSTEM

Empyema: Simple, Interrupted and Continuous Aspiration.—H. M. Richter.
Arch. Pediat., 1917, **34,** 696.

Pneumonias, Unresolved: What Becomes of Them? J. H. Hess.
Arch. Pediat., 1917, **34,** 686.

BLOOD AND CIRCULATORY SYSTEM

Anemia, Pernicious, in Boy of Eight Years.—J. L. Morse and S. B. Wohlbach.
Am. Jour. Dis. Child., 1917, **14,** 301.

Pericarditis Complicating Pneumonia in Infant, Case of.—E. A. Tivey.
Med. Jour. Australia, 1917, **2,** 227.

Tachycardia, Paroxysmal, in Childhood.—N. W. Brown.
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NERVOUS SYSTEM

Brain Development of Child, Influence of Labor on.—M. P. E. Groszmann.
New York Med. Jour., 1917, **106,** 827.

Chorea, Autoserum Treatment of (Goodman); Report of Cases.—E. M. Tarr.
Northwest Med., 1917, **16,** 308.

Cretinism, Nervous.—F. G. Crookshank.
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Delinquents, Female Juvenile, Mental Condition of.—J. H. W. Rhein.
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Little's Disease, Operative Treatment in.—A. F. de Magalhães.
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Nervous Diseases, Bringing Up of Children and.—A. Stern.
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Paralysis, Transient, of Arm, with Pain, in Young Children.—V. Conterno.
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GENITO-URINARY SYSTEM

Calculi, Functioning of Kidneys Containing.—A. P. Martin.
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Circumcision, Bloodless, Cosmetically Perfect.—H. J. Millstone.
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Nephritis of Children, Studies in.—L. W. Hill.
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Pyelitis, Acute, in Children.—C. de Rezende.
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Pyelitis in Children.—H. W. Long.
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Pyelonephritis, Experimental, in Rabbit, Effect of Chilling on.—H. F. Helmholz and B. Rappaport.
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Urine, New Apparatus for Collection of, in Female Infants.—H. O. Ruh.
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SKIN AND APPENDAGES

Erythrodermic Congenitale Ichthyosiforme; Report of Cases and Review of Literature.—G. M. MacKee and I. Rosen.
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Perlèche; Review of Literature. J. E. Lane.
Jour. Cutan. Dis., 1917, **35,** 433.

EYE, EAR, NOSE AND THROAT

Adenoids and Tonsils.—L. R. H. Gantt.
Southern Med. Jour., 1917, **10,** 818.

Blind, Need for More Thorough Examination into Family History and More Exhaustive Search for Disease in Applicants for Admission to Schools for.—J. W. Charles and H. D. Lamb.
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Dacryocystitis, Treatment of, in Infants.—J. Green, Jr.
Missouri State Med. Assn. Jour., 1917, **14,** 427.

Eyes of New-Born, Inflammation of; Observations of Operation of Present Law.—J. E. Bauman.
Ohio State Med. Jour., 1917, **13,** 656.

Otitis Media, Chronic Secretory, Changing Methods and Advances in Treatment of Progressive Deafness from.—F. P. Emerson.
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Otitis Media, Postgrippal.—J. L. Jones.
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Tonsillar Surgery, Intelligent Plea for.—C. H. Long.
Michigan State Med. Soc. Jour., 1917, **16,** 436.

Tonsillectome, Beck-Pierce, Experiences with.—H. Dupuy.
Laryngoscope, 1917, **27,** 769.

Tonsillectomy, Hemorrhage Following.—H. H. Amsden.
Boston Med. and Surg. Jour., 1917, **177,** 594.

SURGERY AND ORTHOPEDICS

Harelip and Cleft Palate, Operations for.—W. C. Gewin.
Med. Rec., New York, 1917, **92,** 679.

Scoliosis, Treatment of, Remote Results of.—A. Serra and G. Valtancoli.
Chir. d. org. di mov., Bologna, 1917, **1,** 380.

THERAPEUTICS

Heliotherapy in Great Britain.—C. E. Warner.
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MISCELLANEOUS

Babies, Scientific.—H. B. Wentz.
Journal-Lancet, 1917, **37,** 641.

Births and Deaths in Metropolitan Areas, Enforcement of Registration of, History, Methods, Results and Checks.—W. H. Guilfoy.
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Children's Diseases, Civilization and.—R. G. Freeman.
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Children, Welfare Work for, at Havre During the War.—A. Loir and H. Legangneux.
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Clothing, Weight of, and Its Relation to Weight of Child in First Five Years of Life.—J. P. Crozer Griffith.
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Deformity, Congenital, of Leg, Toes and Fingers, Case of.—A. G. Nichol and R. H. Perry.
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Depopulation Problem.—C. Richet.
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Depopulation Problem. Committee Report.—A. Pinard.
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Edema of Right Arm, a Case of Chronic Congenital.—L. Rimbaud and H. Roger.
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Feebleminded as Element in Poverty.—R. W. Kelso.
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Feebleminded Deaf, Plea for Instruction and After-School Care of.—C. A. Yale.
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Feebleminded, Dr. Samuel G. Howe and Beginning of Work for, in Massachusetts.—F. E. Williams.
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Feebleminded, Noncommitted, What It Means to Have Them in Community.—C. C. Carstens.
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Glucose Injections, Intravenous, in Infancy.—C. H. Dunn.
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Growth, Effect of Prolongation of Period of, on Total Duration of Life.—J. H. Northrop.
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Illegitimate Child.—J. J. Kelso.
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Infant Mortality, Avoidable, in Italy.—G. B. Allaria.
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Infant Mortality During the War, Increased, and Means to Combat.—M. Flamini.
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Infant Welfare Work in War Time.—G. L. Meigs.
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Lip Reading Courses, Training Teachers for.—Nager.
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Mentally Deficient Children, Society's Duty to.—D. G. Griffiths.
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Oral Prophylaxis at School Age.—H. W. Guthrie.
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Oral Sepsis from Viewpoint of Pediatrist.—E. M. Tarr.
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Prenatal and Postnatal Work, Relations of.—M. M. Davis.
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Public Health Work of Children's Hospital.—C. V. Dorwarth.
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Quarantines, Why They Fail in Protection of Public Health.—S. A. Woolsey.
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Schoolchildren, Mental and Physical Survey of Retarded.—R. H. Perry.
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School Teachers, How They Can Benefit Physicians.—E. McKendrie.
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Spinal Cavity, Capillary Drainage of.—R. A. Gutmann.
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Urticaria.—O. Joerg.
New York Med. Jour., 1917, **106,** 647.

INDEX TO VOLUME XIV

PAGE

Abt, Isaac A.: Congenital skin defects..... 113

Acid intoxication, postoperative. P. C. Jeans and Meredith R. Johnston.. 57

Age of fetus, diagnosis of, by use of roentgenograms. Julius H. Hess.... 397

Anemia, pernicious, in boy of eight years, case of. John Lovett Morse and S. Burt Wohlbach..... 301

Anemia, secondary, treatment of, in infants, by blood transfusion. Charles Gilmore Kerley..... 470

Bacillus abortus bovinus, observations of presence of, in certified milk. E. C. Fleischner and K. F. Meyer..... 157

Bacteria, intestinal, some observations on, in children. W. W. Ford, K. D. Blackfan and M. D. Batchelor..... 354

Banana as a food for children. Marshall C. Pease and Anton R. Rose.... 379

Bartlett, Frederick H.: Some analyses of vegetables showing the effect of the method of cooking..... 34

Batchelor, M. D.: Some observations on intestinal bacteria in children.... 354

Beeler, Carol: Focal lesions produced in rabbit by colon bacilli isolated from pyelocystitis cases..... 5

Blackfan, Kenneth D.: Internal hydrocephalus. Second paper..... 424

Blackfan, K. D.: Some observations on intestinal bacteria in children..... 354

Blood solids, total variations in infants of, and the concentration of sodium chlorid in the plasma. Angelia M. Courtney and Helen L. Fales..... 202

Blood transfusion, treatment of secondary anemia in infants by. Charles Gilmore Kerley..... 470

Brown, N. Worth: Paroxysmal tachycardia in childhood..... 287

Child life, protection of, during war; editorial..... 1

Cod liver oil, effect of, on growth in a case of intestinal infantilism. L. Emmett Holt, Angelia M. Courtney and Helen L. Fales..... 222

Colon bacilli isolated from pyelocystitis cases, focal lesions produced in rabbit by. Henry F. Helmholz and Carol Beeler..... 5

Conrad, Henry B.: Case of pneumonia of unusually short duration..... 296

Courtney, Angelia M.: Effect of cod liver oil on growth in case of intestinal infantilism..... 222

Courtney, Angelia M.: Some analyses of vegetables showing the effect of the method of cooking..... 34

Courtney, Angelia M.: Variations in infants of total blood solids and the concentration of sodium chlorid in the plasma..... 202

Current pediatric literature.....65, 149, 228, 316, 391, 473

Dandy, Walter E.: Internal hydrocephalus. Second paper..... 424

Delinquents, juvenile, a mental and physical survey of a group of, with notes on the Yerkes-Bridges Point Scale. Harold Kniest Faber and Arthur John Ritter..... 444

Dunn, Charles Hunter: Intravenous glucose injections in infancy..... 52

Eddy, Walter H.: Use of pancreatic vitamin in cases of marasmus..... 189

Energy requirements, total, of infant, twenty-four hour metabolism of two normal infants with special reference to. Fritz B. Talbot..... 25

PAGE

Faber, Harold Kniest: A mental and physical survey of a group of juvenile delinquents, with notes on the Yerkes-Bridges Point Scale..... 444

Fales, Helen L.: Effect of cod liver oil on growth in case of intestinal infantilism 222

Fales, Helen L.: Some analyses of vegetables showing the effect of the method of cooking..... 34

Fales, Helen L.: Variations in infants of total blood solids and the concentration of sodium chlorid in the plasma..... 202

Fetus, age of, diagnosis of, by use of roentgenograms. Julius H. Hess.... 397

Fleischner, E. C.: Observations on presence of bacillus abortus bovinus in certified milk..... 157

Focal lesions produced in rabbit by colon bacilli isolated from pyelocystitis cases. Henry F. Helmholz and Carol Beeler..... 5

Ford, W. W.: Some observations on intestinal bacteria in children..... 354

Friedlander, Alfred: Enlargement of the thymus treated by the roentgen ray 40

Gastric juice in infants' stomachs, hunger and appetite secretion of; Rood Taylor 258

Gliosarcoma in infant of seven weeks resembling hydrocephalus. L. Emmett Holt..... 219

Glucose injections, intravenous, in infancy. Charles Hunter Dunn..... 52

Grulee, C. G.: Rumination in the first year of life..... 210

Helmholz, Henry F.: Focal lesions produced in the rabbit by colon bacilli isolated from pyelocystitis cases..... 5

Hess, Alfred F.: Infantile scurvy; V. A study of its pathogenesis..... 337

Hess, Julius H.: The diagnosis of the age of the fetus by the use of roentgenograms 397

Hill, Lewis Webb: Studies in nephritis of children..... 267

Holt, L. Emmett: Effect of cod liver oil on growth in case of intestinal infantilism 222

Holt, L. Emmett: Gliosarcoma in an infant of seven weeks resembling hydrocephalus 219

Hoobler, B. Raymond: Effect on human milk production of diets containing various forms and quantities of protein..... 105

Huenekens, E. J.: Prophylactic use of pertussis vaccine controlled by complement fixation test..... 283

Hunger and appetite secretion of gastric juice in infants' stomachs. Rood Taylor..... 258

Hunger in infant. Rood Taylor..... 233

Hydrocephalus, internal. Second paper. Walter E. Dandy and Kenneth D. Blackfan..... 424

Infant welfare work in war time. Grace L. Meigs..... 80

Infantilism, intestinal, effect of cod liver oil on growth in case of..... 222

Intestinal infantilism, effect of cod liver oil on growth in case of. L. Emmett Holt, Angelia M. Courtney and Helen L. Fales..... 222

Intussusception of bowels in an infant, followed by obstruction of the bowels with a second intussusception within three weeks after the first operation. A. Peskind..... 63

Jeans, P. C.: Postoperative acid intoxication..... 57

Johnston, Meredith R.: Postoperative acid intoxication..... 57

Kerley, Charles Gilmore: The treatment of secondary anemia in infants by blood transfusion..... 470

PAGE

Kingsbury, F. B.: Uric acid content of the blood in the new-born..... 98
Knox, J. H. Mason, Jr.: Value of the von Pirquet test as controlled by necropsy findings..... 47
Lewis, Fielding O.: Progress in pediatrics. Recent progress in otologic diseases of children..... 307
Marasmus, use of pancreatic vitamin in cases of. Walter H. Eddy and Joseph C. Roper..... 189
Meigs, Grace L.: Infant welfare work in war time..... 80
Metabolism, twenty-four hour, of two normal infants with special reference to the total energy requirements of infants. F. B. Talbot..... 25
Meyer, K. F.: Observations on presence of bacillus abortus bovinus in certified milk 157
Milk, certified, observations on the presence of bacillus abortus bovinus in. E. C. Fleischner and K. F. Meyer..... 157
Milk production, human, effect on, of diets containing various forms and quantities of protein. B. Raymond Hoobler..... 105
Morse, John Lovett: Case of pernicious anemia in boy of eight years..... 301
Nephritis of children, studies in. First paper. Lewis Webb Hill..... 267
New-born, uric acid content of blood in. J. P. Sedgwick and F. B. Kingsbury 98
Nicoll, Matthias, Jr.: Epidemiologic data in the poliomyelitis epidemic in New York State..... 69
Otologic diseases of children, recent progress in. Progress in pediatrics. Fielding O. Lewis..... 307
Pancreatic vitamin, use of, in cases of marasmus. Walter H. Eddy and Joseph C. Roper..... 189
Pease, Marshall C.: Banana as a food for children..... 379
Pediatric literature, current.....65, 149, 228, 316, 391, 473
Pediatrics, progress in.....122, 307
Pertussis vaccine, prophylactic use of, controlled by complement fixation test; E. J. Huenekens..... 283
Peskind, A.: Intussusception of the bowel in an infant, followed by obstruction of the bowels with a second intussusception within three weeks after the first operation..... 63
Pneumonia of unusually short duration, case of. Henry B. Conrad..... 296
Point-Scale, Yerkes-Bridges, a mental and physical survey of a group of juvenile delinquents. Harold Kniest Faber and Arthur John Ritter.. 444
Poliomyelitis, condition of the reflexes in. Joseph G. Regan..... 321
Poliomyelitis epidemic in New York State, epidemiologic data in. Matthias Nicoll, Jr..... 69
Poliomyelitis, review of. Progress in pediatrics. A. B. Schwartz..... 122
Progress in pediatrics.....122, 307
Protein, effect on human milk production of diets containing various forms and quantities of. B. Raymond Hoobler..... 105
Pyelocystitis cases, focal lesions produced in rabbit by colon bacilli isolated from. Henry F. Helmholz and Carol Beeler..... 5
Reflexes, condition of, in poliomyelitis. Joseph G. Regan..... 321
Regan, Joseph G.: Condition of the reflex in poliomyelitis..... 321
Ritter, Arthur John: A mental and physical survey of a group of juvenile delinquents, with notes on the Yerkes-Bridges point scale..... 444

PAGE

Roentgenograms, diagnosis of the age of the fetus by the use of. Julius H. Hess 397
Roper, Joseph C.: Use of pancreatic vitamin in cases of marasmus 189
Rose, Anton B.: The banana as a food for children 379
Rumination in the first year of life. C. G. Grulee 210
Schwartz, A. B.: Review of poliomyelitis. Progress in pediatrics 122
Scurvy, infantile; V. A study of its pathogenesis. Alfred F. Hess 337
Sedgwick, J. P.: Uric acid content of the blood in the new-born 98
Skin defects, congenital. Isaac A. Abt 113
Sodium chlorid in the plasma, concentration of, variations in infants of total blood solids and. Angelia M. Courtney and Helen L. Fales 202
Status lymphaticus, cause of sudden death in. Douglas Symmers 463
Symmers, Douglas: The cause of sudden death in status lymphaticus 463
Tachycardia, paroxysmal, in childhood. N. Worth Brown 287
Talbot, Fritz B.: Twenty-four hour metabolism of two normal infants with special reference to total energy requirements of infants 25
Taylor, Rood: Hunger and appetite secretion of gastric juice in infants' stomachs 258
Taylor, Rood: Hunger in infant 233
Thymus, enlargement of, treated by Roentgen ray. Alfred Friedlander ... 40
Torres, Calixto: The relation of the reaction of the urine to the diet in infants and children 365
Uric acid content of blood in new-born. J. P. Sedgwick and F. B. Kingsbury 98
Urine, relation of the reaction of, to diet in infants and children. Calixto Torres 365
Vegetables, some analyses of, showing effect of method of cooking. Angelia M. Courtney, Helen L. Fales and Frederic H. Bartlett 34
Von Pirquet test, value of, as controlled by necropsy findings. J. H. Mason Knox, Jr 47
Wohlbach, S. Burt: Case of pernicious anemia in boy of eight years 301
Yerkes-Bridges point scale, notes on, A mental and physical survey of a group of juvenile delinquents. Harold Kniest Faber and Arthur John Ritter 444

CONTENTS OF VOLUME XIV

OCTOBER, 1917. NUMBER 4

PAGE

HUNGER IN THE INFANT. ROOD TAYLOR, M.D., Sc.D., ROCHESTER, MINN.... 233

HUNGER AND APPETITE SECRETION OF GASTRIC JUICE IN INFANTS' STOMACHS. ROOD TAYLOR, M.D., Sc.D., ROCHESTER, MINN..... 258

STUDIES IN THE NEPHRITIS OF CHILDREN. FIRST PAPER. LEWIS WEBB HILL, M.D., BOSTON..... 267

THE PROPHYLACTIC USE OF PERTUSSIS VACCINE CONTROLLED BY THE COMPLEMENT FIXATION TEST. E. J. HUENEKENS, M.D., MINNEAPOLIS..... 283

PAROXYSMAL TACHYCARDIA IN CHILDHOOD. N. WORTH BROWN, M.D., BALTIMORE..... 287

CLINICAL DEPARTMENT:

A CASE OF PNEUMONIA OF UNUSUALLY SHORT DURATION. HENRY B. CONRAD, M.D., BALTIMORE..... 296

CASE OF PERNICIOUS ANEMIA IN A BOY OF EIGHT YEARS. JOHN LOVETT MORSE, A.M., M.D., AND S. BURT WOHLBACH, M.D., BOSTON..... 301

PROGRESS IN PEDIATRICS:

RECENT PROGRESS IN OTOLOGIC DISEASES OF CHILDREN. FIELDING O. LEWIS, M.D., PHILADELPHIA..... 307

CURRENT PEDIATRIC LITERATURE..... 316

NOVEMBER, 1917. NUMBER 5

PAGE

THE CONDITION OF THE REFLEXES IN POLIOMYELITIS. JOSEPH G. REGAN, M.D., BROOKLYN..... 321

INFANTILE SCURVY. V. A STUDY OF ITS PATHOGENESIS. ALFRED F. HESS, M.D., NEW YORK..... 337

SOME OBSERVATIONS ON INTESTINAL BACTERIA IN CHILDREN. W. W. FORD, K. D. BLACKFAN AND M. D. BATCHELOR, BALTIMORE..... 354

THE RELATION OF THE REACTION OF THE URINE TO THE DIET IN INFANTS AND CHILDREN. CALIXTO TORRES, M.D., BOGOTA, COLOMBIA, S. A..... 365

THE BANANA AS A FOOD FOR CHILDREN. MARSHALL C. PEASE, M.D., AND ANTON R. ROSE, M.S., PH.D., NEW YORK..... 379

PROGRESS IN PEDIATRICS:

CURRENT PEDIATRIC LITERATURE..... 391

CONTENTS

PAGE

THE DIAGNOSIS OF THE AGE OF THE FETUS BY THE USE OF ROENTGENOGRAMS. JULIUS H. HESS, M.D., CHICAGO 397

INTERNAL HYDROCEPHALUS. SECOND PAPER. WALTER E. DANDY, M.D., AND KENNETH D. BLACKFAN, M.D., BALTIMORE 424

A MENTAL AND PHYSICAL SURVEY OF A GROUP OF JUVENILE DELINQUENTS. WITH NOTES ON THE YERKES-BRIDGES POINT SCALE. HAROLD KNIEST FABER, M.D., AND ARTHUR JOHN RITTER, SAN FRANCISCO 444

THE CAUSE OF SUDDEN DEATH IN STATUS LYMPHATICUS. DOUGLAS SYMMERS, M.D., NEW YORK 463

THE TREATMENT OF SECONDARY ANEMIA IN INFANTS BY BLOOD TRANSFUSION. CHARLES GILMORE KERLEY, M.D., NEW YORK 470

PROGRESS IN PEDIATRICS:
CURRENT PEDIATRIC LITERATURE 473

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